Human Space Colonisation and Endosymbiotic Archaeal Colonies

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Chapter 1

Human Metabolonomics, Symbiotic Evolution and Space Colonisation

Human beings can be re-engineered to live in an anaerobic environment. This is possible by membrane sodium potassium ATPase inhibition and sodium potassium ATPase mediated ATP synthesis and by anaerobic glycolysis. Low level of EMF and photons can also mediate ATP synthesis via the electron transport chain. This occurs by inducing high level of archaeal symbiosis. This can be achieved by feeding low fibre diet, fecal transplantation and archaeal infusions. The archaea growth is increased in the gut and undergoes endosymbiosis generating new organelle - fructosoid, neurotransminoid, steroidelle, porphyrinoid, transmutoid and glycosaminoglycoid. This changes the human species to survive in an anaerobic environment or in an environment of ammonia or hydrogen sulphide. Archaeal symbiosis produces neanderthalisation of human beings which can survive in extremophilic environments including outside planetary system. The human systems exist as a habitat for an ecology of symbiotic bacteria and viruses. The human genes are small microislands floating in an ocean of bacterial and viral genes. The question of human body survival in extremophilic conditions of other planets can be solved by considering the survival of the symbiotic bacteria and viruses of the human systems. The human systems exist for the thriving and growth of the symbiotic bacteria and viruses. The induction of colonic and endosymbiotic archaeal symbiosis will induce changes in the ecosystem of endosymbiotic archaea, gut archaea, human tissues and archaeal phages. Metabolic engineering can be done for the survival of this ecosystem with human zombies functioning as the habitat for extremophilic symbiotic archaea. This is the crux for the survival of human beings in outer planetary system and establishing human colonies in outer planetary systems. This holds the key for human survival as a species and a race.

Space colonisation involves human engineering. This requires archaeal symbiosis and shutting down of the human system. The archaeal network takes

over. The human being is converted into a zombie. This is done by colonic infusion, i/v infusion of archaea and consuming a low fibre diet. This conditions the body to exist in an anaerobic environment.

The archaeal symbiosis creates a new human species, the neoneanderthals. It has a dominant cerebellar function and is impulsive, creative and autistic. It is artistic, musical and aesthetic. It is intuitive and capable of extrasensory perception and telepathy. The species can communicate with computers and modulate their programs by interaction with the archaeal colony network which is capable of quantal perception. The archaeal colony network of magnetite is capable of quantal perception and can be called as a conscious artificial intelligence. The archaeal colony network via quantal perception can have two-way communication with computer programs producing a synthestic brain.

This produces what is called as a quantal civilization. The porphyrions can emerge out of the quantal foam like the lotus of brahma. The porphyrions are molecules which can have a wave-particle existence and the porphyrions forms a template for RNA viroids, DNA viroids and prions to form. They symbiose to form the archaea. The archaea has an abiogenic replication. Consciousness is mediated by gravity and the unconscious world is mediated by anti-gravity acting via archaeal colony network. Thought creates matter. The world of matter is embedded in thought. The universe and anti-universe as well as parallel universes are created out of the phenomena of soni-luminescence arising out of gravitational and anti-gravitational waves. The mind creates the universe. This is exemplified by the concept of the universe arising out of the word *Om* in Hindu philosophy. This forms the basis of human colonisation of other planetary systems.

The archaeal colony network can function on the basis of zero point energy derived from gravitational waves. The archaeal colony network is capable of quantal perception. The archaeal colonies are capable of nuclear fusion and

fission. This can generate energy for the human colonies in other planetary system. Thus human colonies in other planetary system are a possibility based on metabolic human engineering created by archaeal symbiosis.

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[1] Kurup, R. K. and Kurup, P. A. Global Warming, Archaea and Viroid Induced Symbiotic Human Evolution - The Atavistic Neoneanderthalisation and Gut Microflora - Archaeal RNA Viroidal Quasi-Species Consortia and Human Species Identity. New York: Open Science Publishers, 2016.

Chapter 2

Human Space Colonisation and Human Metabolic Engineering - Human Neanderthalic Metabolonomics - Generation of New Organelle

Introduction

Human beings can be metabolically re-engineered to live in an anaerobic environment as occurs in space colonization. This is possible by membrane sodium potassium ATPase inhibition and sodium potassium ATPase mediated ATP synthesis and by anaerobic glycolysis. Low level of EMF and photons can also mediate ATP synthesis via the electron transport chain. This occurs by inducing high level of archaeal symbiosis. This can be achieved by feeding low fibre diet, fecal transplantation and archaeal infusions. The archaea growth is increased in the gut and undergoes endosymbiosis generating new organelle - fructosoid, neurotransminoid, steroidelle, porphyrinoid, transmutoid and glycosaminoglycoid. This changes the human species to survive in an anaerobic environment or in an environment of ammonia or hydrogen sulphide. Archaeal symbiosis produces neanderthalisation of human beings which can survive in extremophilic environments including outside planetary system. The human systems exist as a habitat for ecology of symbiotic bacteria and viruses. The human genes are small microislands floating in an ocean of bacterial and viral genes. The question of human body survival in extremophilic conditions of other planets can be solved by considering the survival of the symbiotic bacteria and viruses of the human systems. The human systems exist for the thriving and growth of the symbiotic bacteria and viruses. The induction of colonic and endosymbiotic archaeal symbiosis will induce changes in the ecosystem of endosymbiotic archaea, gut archaea, human tissues and archaeal phages. Metabolic engineering can be done for the survival of this ecosystem with human zombies functioning as the habitat for extremophilic symbiotic archaea. This is the crux for the survival of human beings in outer planetary system and establishing human colonies in outer planetary systems. This holds the key for human survival as a species and a race. Archaeal symbiosis leads to

neanderthalisation of the homo sapien species. This can be described as symbiosis mediated evolution. The symbiotic archaea catabolise cholesterol and generate the steroidal glycosidic hormone digoxin. Neanderthal metabolonomics is primarily mediated by archaeal metabolonomics and archaeal symbiosis. They have got cholesterol catabolism, the shikimic acid pathway, more of anaerobic glycolysis, increase connective tissue synthesis, fructolysis, nucleic acid synthesis and mitochondrial dysfunction. The homo neanderthalis has different personality and social characteristics with increased creative, gender equal, matriarchal, asexual and alternate sexual, spiritual, intuitive, surrealistic and community centered characteristics. Archaeal symbiosis endosymbiotically and in the colonic microflora can produce neanderthalisation. The Neanderthal phenotype is blood cytochrome F420 positive. The homo sapien phenotype has less of archaeal symbiosis endosymbiotic and colonic. The homo sapien phenotype cytochrome F420 negative. The homo sapien species is less creative, patriarchal, gender unequal, heterosexual, logical and individualistic. Homo sapien metabolonomics is primarily aerobic and mitochondrial. They consume a high fibre diet which generates colonic butyrate and strengthens the gut blood and blood brain barrier limiting endosymbiotic archaeal growth. The blood archaeal cytochrome F420 a marker of endosymbiotic archaeal growth is reduced in homo sapiens as well as the levels of endogenous digoxin generated by cholesterol catabolism. The homo sapiens can be adapted to anaerobic environment in other planetary systems by metabolic engineering induced by consumption of colonic microflora probiotic from normal neanderthalic anaerobic adapted phenotypes who are blood cytochrome F420 positive. This can be supplemented by taking a low fibre, high protein high fat diet which will increase the colonic gut archaea. The low fibre high protein high fat diet will also reduce the gut butyrate which will break the gut blood barrier and blood brain barrier producing increased

endosymbiotic archaeal growth. The species change is a gut microflora and endosymbiotic flora mediated change which can be termed as induced evolution. Induction of species change between aerobic metabolism adapted homo sapiens and anaerobic metabolism homo neanderthalis was induced by feeding colonic microflora from neanderthalic phenotype which is cytochrome F420 positive. The aerobic metabolism adapted homo sapien species is converted to anaerobic metabolism adapted Neanderthal species by giving a high fat, high protein diet derived from black gram and coconut. The homo sapien flora can induce conversion of anaerobic metabolism adapted neanderthalis to aerobic metabolism adapted homo sapien species and the neanderthalis flora can induce conversion of aerobic metabolism adapted sapiens to anaerobic metabolism adapted neanderthalis species. A high fibre diet induces neanderthalisation and anaerobic metabolism adaption. A low fibre diet produces homo sapienisation and aerobic metabolism adaption. This patent is for the modulation of endosymbiotic archaeal growth and endogenous digoxin synthesis resulting in phenotypic metabolonomic and genotypic change in human species from homo sapiens to homo neanderthalis as well as metabolic engineering for survival in anaerobic environments of other planetary systems.

Research Work Carried Out

The homo sapiens can be adapted to anaerobic environment in other planetary systems by metabolic engineering induced by consumption of colonic microflora probiotic from normal neanderthalic anaerobic adapted phenotypes who are blood cytochrome F420 positive. This can be supplemented by taking a low fibre, high protein high fat diet which will increase the colonic gut archaea. The low fibre high protein high fat diet will also reduce the gut butyrate which will break the gut blood barrier and blood brain barrier producing increased endosymbiotic archaeal growth. The species change is a gut microflora and

endosymbiotic flora mediated change which can be termed as induced evolution. Induction of species change between aerobic metabolism adapted homo sapiens and anaerobic metabolism homo neanderthalis was induced by feeding colonic microflora from neanderthalic phenotype which is cytochrome F420 positive. The aerobic metabolism adapted homo sapien species is converted to anaerobic metabolism adapted Neanderthal species by giving a high fat, high protein diet derived from black gram and coconut. The homo sapien flora can induce conversion of anaerobic metabolism adapted neanderthalis to aerobic metabolism adapted homo sapien species and the neanderthalis flora can induce conversion of aerobic metabolism adapted sapiens to anaerobic metabolism adapted neanderthalis species. A high fibre diet induces neanderthalisation and anaerobic metabolism adaption. A low fibre diet produces homo sapienisation and aerobic metabolism adaption. This patent is for the modulation of endosymbiotic archaeal growth and endogenous digoxin synthesis resulting in phenotypic metabolonomic and genotypic change in human species from homo sapiens to homo neanderthalis as well as metabolic engineering for survival in anaerobic environments of other planetary systems. The decreased in archaeal density in the colonic microbiota and endosymbiotic archaeal community leads to moderately increased digoxin synthesis and the acquired immunodeficiency syndrome. A high fibre diet can lead to decrease in archaeal colonic density and decrease in endosymbiotic archaea. A high fibre diet produces increased butyrate which strengthens the gut blood and brain blood barrier leading to decreased endosymbiotic archaeal density. A low fibre diet leads to increased density of colonic archaeal population and decreases the clostridial clusters generating butyrate. The deficiency of butyrate leads to breaching of the blood brain barrier and gut blood barrier producing increased endosymbiotic archaeal growth. The increase in endosymbiotic archaeal growth in populations consuming a low fibre diet leads to large increases in digoxin producing a

hyperdigoxinemic state. In low fibre diet consuming population the endosymbiotic archaeal density is also very high. The low microflora butyrate induced HDAC inhibition contributes to reduced HERV expression and reduction in size of the cerebral cortex and a dominant cerebellar function. This produces a neanderthalised brain and phenotype in populations on a low fibre diet. The reduced HERV expression in neanderthalised phenotype contributes to a cerebellar dominant brain and increased HERV expression in the homo sapien phenotype contributes to a cerebellar cortical dominant brain.

Endogenous actinidic archaea have been detected in homo neanderthalis by increased blood cytochrome F420 activity. They can survive in anaerobic environments. The homo sapien species have less of archaeal symbiosis and are blood cytochrome F420 negative. They can survive in aerobic environments. The archaea are detected by spectrophotometry for cytochrome F420, the methanogenic cytochrome in the blood. The endogenous actinidic archaea synthesizes cholesterol by the mevalonate pathway. The cholesterol is catabolized to digoxin. Cytochrome F420 activity in the blood determines the homo neanderthalis species and lack of cytochrome F420 activity in the blood determines the homo sapien species. The homo neanderthalis has different personality and social characteristics with increased creative, gender equal, matriarchal, asexual and alternate sexual, spiritual, intuitive, surrealistic and community centered characteristics. The homo sapien species is less creative, patriarchal, gender unequal, heterosexual, logical and individualistic. Neanderthal metabolonomics is primarily mediated by archaeal metabolonomics and archaeal symbiosis. They have got cholesterol catabolism, the shikimic acid pathway, more of anerobic glycolysis, increase connective tissue synthesis, fructolysis, nucleic acid synthesis and mitochondrial dysfunction. Homo sapien metabolonomics is primarily aerobic and

mitochondrial. The species change is a gut microflora and endosymbiotic flora mediated change which can be termed as induced evolution.

Metabolic Engineering for Species Change for Space Colonisation and Human Survival in Anaerobic Conditions Occurring in Space Colonization - Colonic Flora Probiotic Administration to Aerobic Adapted Blood Cytochrome F420 Negative Homo Sapiens from Anaerobic Adapted Blood Cytochrome F420 Positive Homo Neanderthalis

Research work carried out by us over a period of years has shown patients have this disorders or condition show a significant improvement on the natural organic paleo probiotic when endogenous archaeal growth and digoxin synthesis is demonstrated in the patients. Populations are screened for endosymbiotic archaeal activity in the sera by analysis of cytochrome F420 activity. The population that is positive for cytochrome F420 activity is chosen for the collection of the specimen. The blood cytochrome F420 positive population was taken as homo neanderthalis phenotype. The population was fed on a paleo diet of high dietary fat coconut oil medium chain triglyceride and pulse/legume protein. The normal fecal collection was done from a healthy normal genetically related individual chosen by the patient and the administration of the organic natural probiotic isolated from the genetically related individual was volitional and a patient decision. The permission of the Ethics Committee of the Institute - Metabolic Disorders Research Centre, Trivandrum was obtained. The fresh fecal matter from healthy humans was collected. Around 100 g of the organic matter is used in the preparation of the product. 100 g of the organic matter is diluted with normal saline and centrifuged at 2500 rpm. The rough matter forms a deposit and the supernatant is collected. The supernatant is preserved by adding 25 g of trehalose which can preserve the probiotic bacteria. This supernatant with added trehalose is freeze-dried and packed in double gelatin capsules. This capsule can be

administered orally. The population with homo sapien characteristics was given fecal colonic flora preparation from neanderthalic phenotypes in the manner described above. The anaerobic adapted neanderthalic phenotypes were cytochrome F420 positive in their blood. Thus interconversion of species was possible by administration of probiotic from colonic flora of homo sapiens and homo neanderthalis identified by cytochrome F420 activity in blood. The homo neanderthalis species can survive in the anaerobic environment of other planetary system.

Metabolic Engineering for Species Change for Anaerobic Adaptation Occurring in Space Colonisation - Low Fibre High Fat High Protein Diet to Produce Neanderthalisation Which Is Anaerobic Adapted for Space Colonisation

High archaeal growth induces neanderthalisation of human species. Feeding the homo sapiens a low fibre high fat high protein paleo diet from coconut oil and pulse protein will increase the endosymbiotic archaeal growth and colonic archaeal microbiota. The increase in archaeal density will increase digoxin synthesis. This will help to neanderthalise the homo sapien species and make them anaerobic adaptable. This was done by giving 100 g of medium chain triglyceride from coconut oil combined with 100 g of black gram pulse protein. This helps to convert the homo sapien phenotype to Neanderthal phenotype which are anaerobic adapted. The low fibre diet will increase gut archaeal growth. The gut butyrate also increases breaching the gut blood barrier increasing endosymbiotic archaeal growth and digoxin synthesis. The homo sapien species when fed a low fibre high fat high protein non-vegetarian diet has increased density of gut archaeal microflora and endosymbiotic archaeal growth. The gut butyrate generation is reduced and the gut blood barrier and blood brain barrier is breached. This leads to increase in endosymbiotic archaea and the homo sapien species gets converted to homo neanderthalis species. The

homo neanderthalis species is adapted to survive in the anaerobic environment of other planetary system.

Details of the Trial for Homo Sapien Anaerobic Adaptation for Space Colonisation

Archaeal symbiosis leads to neanderthalisation of the homo sapien species. This can be described as symbiosis mediated evolution. The homo neanderthalis has different personality and social characteristics with increased creative, gender equal, matriarchal, asexual and alternate sexual, spiritual, intuitive, surrealistic and community centered characteristics. Neanderthal metabolonomics is primarily mediated by archaeal metabolonomics and archaeal symbiosis. They have got cholesterol catabolism, the shikimic acid pathway, more of anaerobic glycolysis, increase connective tissue synthesis, fructolysis, nucleic acid synthesis and mitochondrial dysfunction. The homo sapien species is less creative, patriarchal, gender unequal, heterosexual, logical and individualistic. Homo sapien metabolonomics is primarily aerobic and mitochondrial. The species change is a gut microflora and endosymbiotic flora mediated change which can be termed as induced evolution. The feeding of the homo sapien phenotype with a low fibre high fat high protein vegetarian diet from coconut oil and black gram protein resulted in increased in archaeal density in the gut microflora and endosymbiotic archaeal growth in the blood as measured by cytochrome F420 activity and neanderthalisation of the homo sapien species. This makes the homo sapien species neanderthalised with a different phenotype, genotype and psychological type.

The population were assessed before dietary modulation was started and by all required laboratory investigations. The duration of modulatory regimes ranged from 6 months. Their condition was assessed during treatment and after treatment clinically and using all necessary laboratory investigations. This produced a

change in the aerobic adapted homo sapien phenotype to anaerobic adapted homo neanderthalis phenotype. The homo neanderthalis species is adapted to survive in the anaerobic environment of planetary systems other than earth.

Population Included in the Large Scale Trial to Generate Neanderthalised Species Which Are Anaerobic Adapted for Space Colonisation

The population included in the large-scale trial of homo sapien phenotype identified by lower or absent cytochrome F420 activity in blood. They were fed a low fibre, high fat, high protein, vegetarian diet from coconut oil and black gram protein for 6 months. This resulted in increase in endosymbiotic and colonic archaeal density and neanderthalisation of the homo sapien phenotype. The homo sapien phenotype given colonic microflora capsules from normal Neanderthal phenotypes with high cytochrome F420 activity also resulted in neanderthalisation of homo sapien phenotype. The psychological characteristics changed from homo sapien less creative, patriarchal, gender unequal, heterosexual, logical and individualistic to neanderthalic increased creative, gender equal, matriarchal, asexual and alternate sexual, spiritual, intuitive, surrealistic and community centered characteristics. The metabolic phenotype changed from homo sapien mitochondrial phenotype to neanderthalic cholesterol catabolism, the shikimic acid pathway, more of anaerobic glycolysis, increase connective tissue synthesis, fructolysis, nucleic acid synthesis and mitochondrial dysfunction phenotype.

Conclusion

A method to induce evolutionary changes in the human species by modulating archaeal symbiosis and interconverting aerobic adapted homo sapien to anaerobic adapted homo neanderthalis is described. This is done by a low fibre diet, high protein high fat diet derived from coconut oil and black

gram protein as well as by administering colonic microflora probiotic from human cytochrome F420 positive Neanderthal healthy population. This is a methodology to modulate species interconversion from aerobic adapted homo sapien to anaerobic adapted homo neanderthalis with its attendant changes in psychological, phenotypic and metabolonomic characteristics of the population. This can be called as a therapeutic archaeal symbiotic modulated human evolution. The anaerobic adapted Neanderthal species is more fit to survive space colonization in other planetary settings.

Chapter 3

Endosymbiotic Pathogenic Archaea and Archaeal Derived RNA Viroids Induced Evolutionary Species Change in Humans - Interconversion of Homo Sapiens and Homo Neanderthalis - Method for Archaeal Symbiosis Modulated Human Evolution for Therapeutic Purpose

Introduction

Archaeal symbiosis leads to neanderthalisation of the homo sapien species. This can be described as symbiosis mediated evolution. The homo neoneanderthalis has an increase predilection to metabolic syndrome X, strokes, CAD, hyperlipidemia, diabetes mellitus, autoimmune, neuropsychiatric, neurodegenerative, cancer and are retroviral resistant. The homo neanderthalis has different personality and social characteristics with increased creative, gender equal, matriarchal, asexual and alternate sexual, spiritual, intuitive, surrealistic and community centred characteristics. The homo sapien species are resistant to metabolic syndrome X, strokes, CAD, hyperlipidemia, diabetes mellitus, autoimmune, neuropsychiatric, neurodegenerative, cancer and are retroviral susceptible. The homo sapien species is less creative, patriarchal, gender unequal, heterosexual, logical and individualistic. Neanderthal metabolonomics is primarily mediated by archaeal metabolonomics and archaeal symbiosis. They have got cholesterol catabolism, the shikimic acid pathway, more of anaerobic glycolysis, increase connective tissue synthesis, fructolysis, nucleic acid synthesis and mitochondrial dysfunction. Homo sapien metabolonomics is primarily aerobic and mitochondrial. The species change is a gut microflora and endosymbiotic flora mediated change which can be termed as induced evolution. Induction of species change between homo sapiens and homo neanderthalis was induced by feeding (1) a natural organic probiotic from human colonic flora homo sapiens flora versus neanderthalis flora depending upon phenotypic characteristics. The homo sapien flora can induce conversion of neanderthalis to sapien species and the neanderthalis flora can induce conversion of sapiens to neanderthalis species (2) a new paleo high fibre, high medium chain triglyceride, high legume protein ketogenic diet versus a high fat high protein diet. The high fibre high MCT high legume protein ketogenic diet

converts the neanderthalis to sapien species and a low fibre high protein high fat diet converts the sapien species to neanderthalis species (3) a natural organic probiotic from dung of the Indian cow, Bos primigenius which converts the neanderthalis to homo sapien phenotype (4) a natural antioxidant antibiotics derived from crude extracts Curcuma longa, Moringa pterygosperma, Emblica officinalis, Zingiber officinale, Allium sativum and Withania somnifera for modulation of endosymbiotic archaeal growth and endogenous digoxin synthesis resulting in phenotypic metabolonomic and genotypic change in human species from homo sapiens to homo neanderthalis. The colonic and endosymbiotic archaea and other microbes like clostridial clusters determine the species, race, caste, community and personal identity of the individual. The identity of the individual - personal, community, caste, race, nationality and species is determined by the colonic and endosymbiotic archaeal and clostridial clusters. Predominant archaeal symbiosis produces homo neanderthalis and less prominent archaeal symbiosis and dominant clostridial clusters in the gut produces the homo sapien species. Each individual, race, nationality, caste, creed and community have the endosymbiotic and colonic microbiota signature. This colonic and endosymbiotic microbiota signature is transferable by the change of endosymbiotic and colonic microbiota from one group to another. Thus the evolution and identity based on individuality, race, nationality, caste and creed can be induced.

The research work carried out by us over a period of years showed that patients of these disorders mentioned show:

(1) Decrease in the activity of a cell membrane based enzyme known as sodium potassium ATPase. An inhibition of sodium potassium ATPase produces increase in intracellular calcium and decrease in intracellular magnesium.

(2) Membrane sodium potassium ATPase inhibition is produced by endogenous digoxin which is synthesized from cholesterol by actinidic archaea which acts as endosymbionts in cell. The archaea synthesizes digoxin from cholesterol.

(3) Actinidic archaeal growth has been detected in metabolic syndrome X, coronary artery diseases, strokes, diabetes mellitus, hyperlipidemia, autoimmune, neuropsychiatric, neurodegenerative, cancer and infections

(4) The paleo probiotic from human colonic flora are anti-archaeal agents. The paleo probiotic block the archaeal mevolanate pathway. This decreases digoxin synthesis from cholesterol and treats these chronic disorders.

Detection of Endogenous Actinidic Archaea

Endogenous actinidic archaea have been detected in metabolic syndrome X, diabetes mellitus, CAD, stroke, autism, autoimmune, neuropsychiatric, neurodegenerative, cancer and infections. The archaea are detected by spectrophotometry for cytochrome F420, the methanogenic cytochrome in the blood. The endogenous actinidic archaea synthesizes cholesterol by the mevalonate pathway. The cholesterol is catabolized to digoxin. Digoxin inhibits membrane sodium-potassium-ATPase and increases intracellular calcium and depletes magnesium stores in the cell. This leads to metabolic syndrome X, diabetes mellitus, CAD, stroke, autism, autoimmune, neuropsychiatric, neurodegenerative, cancer and infections. The synthesis of digoxin can be demonstrated in patients by adding cholesterol substrate and cerium to patient's serum and checking for the rise in cytochrome F420 activity and digoxin levels. Digoxin levels are assayed by elisa and cytochrome F420 by spectrophotometry. The test is available in the Metabolic Disorders Research Centre. The patient in whom endogenous archaea and digoxin synthesis is demonstrated is given

nutritional dietary supplements to modulate the effects of archaea and digoxin. This helps to ameliorate the chronic diseases like metabolic syndrome X, diabetes mellitus, CAD, stroke, autism, autoimmune, neuropsychiatric, neurodegenerative, cancer and infections. Cytochrome F420 activity in the blood determines the homo neanderthalis species and lack of cytochrome F420 activity in the blood determines the homo sapien species. The homo neoneanderthalis has an increase predilection to metabolic syndrome X, strokes, CAD, hyperlipidemia, diabetes mellitus, autoimmune, neuropsychiatric, neurodegenerative, cancer and are retroviral resistant. The homo neanderthalis has different personality and social characteristics with increased creative, gender equal, matriarchal, asexual and alternate sexual, spiritual, intuitive, surrealistic and community centred characteristics. The homo sapien species are resistant to metabolic syndrome X, strokes, CAD, hyperlipidemia, diabetes mellitus, autoimmune, neuropsychiatric, neurodegenerative, cancer and are retroviral susceptible. The homo sapien species is less creative, patriarchal, gender unequal, heterosexual, logical and individualistic. Neanderthal metabolonomics is primarily mediated by archaeal metabolonomics and archaeal symbiosis. They have got cholesterol catabolism, the shikimic acid pathway, more of anerobic glycolysis, increase connective tissue synthesis, fructolysis, nucleic acid synthesis and mitochondrial dysfunction. Homo sapien metabolonomics is primarily aerobic and mitochondrial. The species change is a gut microflora and endosymbiotic flora mediated change which can be termed as induced evolution.

Main Objectives of the Study

The gut microflora regulates body functions. The microflora modulates the immune system, the neuronal system and endocrine system. Alteration in the gut microflora as well as endosymbiotic bacteria has been related to human disease

and evolution of human species. Increase in archaeal growth has been related to psychiatric disorders, tumours, autoimmune disease, metabolic syndrome and degenerations. The archaea forms a major chunk of the gut microflora. The archaea can leach into the tissue systems forming endosymbionts which can function like cellular organelle and can catabolise cholesterol. The symbiotic archaea can produce a Warburg phenotype and stem cell transformation. This can lead onto human diseases - psychiatric disorders, tumours, autoimmune disease, metabolic syndrome and degenerations. The overgrowth of symbiotic archaea can lead onto change in human species type and create a species with Neanderthal metabolonomics. This disease process leading onto psychiatric disorders, tumours, autoimmune disease, metabolic syndrome and degenerations can be reversed by altering the gut microflora and populating it with non-archaeal phenotypes. This can be done by oral administration of fecal microflora from healthy population.

Symbiosis by microorganisms especially archaea drives the evolution of the species. In such a case symbiosis can be modulated by transfer of microflora symbionts and evolution induced. Endosymbiosis by archaea as well as archaeal symbionts in the gut can modulate the genotype, the phenotype, the social class and the racial group of the individual. The symbiotic archaea can have horizontal and vertical transmission. Endosymbiotic archaeal growth leads to neanderthalisation of the species. The inhibition of the endosymbiotic archaeal growth on the other hand leads to evolution of the homo sapiens. Symbiosis mediated evolution depends on the gut flora and the diet. The combination of the human genome and the symbiotic microbial genome is called the hologenome drives human evolution as well as animal evolution. Endosymbiotic archaeal growth and neanderthalisation can lead to autoimmune disease, metabolic syndrome X, neurodegeneration, cancer, autism and schizophrenia. The Neanderthal gut flora and endosymbiotic archaea was determined by the non vegetarian ketogenic high fat high protein diet consumed by them in the Eurasian

steppes. The homo sapiens including the classical Aryan tribes and African ate a high fibre diet and had lower archaeal growth both endosymbiotic and gut. The dietary fibre intake determines the microbial diversity of the gut. The high fibre intake is associated with increased generation of short chain fatty acids - butyric acid by the gut flora. Butyrate is a HDAC inhibitor and leads to increased generation and incorporation of endogenous retroviral sequences which function as jumping genes. The high dietary fibre intake related increased genomic HERV sequences leads to a dynamic genome, increased synaptic connectivity and a dominant frontal cortex as seen in homo sapien species. The neanderthalic species consume a ketogenic non vegetarian high fat high protein low fibre diet. This leads to decreased generation of endogenous HERV sequences and reduced genomic flexibility in neanderthalic species. This produces smaller cerebral cortex and a dominant cerebellar cortex in the neanderthalic brain. The homo neanderthalic species by the low dietary fibre intake starve their microbial self. This leads to increased endosymbiotic and gut archaeal growth. The mucous membrane lining the gut becomes thinned out as the gut bacteria eats up the mucous lining of the gut. The reduced generation of gut butyrate consequent to increased archaeal growth also damages the gut blood and blood brain barrier. This results in leakage of endotoxins and archaea from the gut to the blood breaching the barrier and produce a chronic immunostimulatory inflammatory state which forms the basis of autoimmune disease, metabolic syndrome, neurodegeneration, oncogenic and psychiatric disorders. The Neanderthal species eat a low fibre diet and have a deficiency of microbiota accessed carbohydrate generating short chain fatty acid. There is a deficiency of butyrate generated in the gut from the dietary fibre which can produce suppression of the chronic inflammatory process. The Neanderthals have got the fermentation by-product deficiency syndrome. The induction of neanderthalic species depends on the low fibre intake induced high archaeal density endosymbiotic and the gut microflora.

The homo sapiens species consume a high fibre diet generating large amounts of short chain fatty acid butyrate which inhibits endosymbiotic and gut archaeal growth. The microbial self of the homo sapien species is more diverse than that of the neanderthalic species and the archaeal population density is less. This results in a protection against chronic inflammation and the induction of diseases like autoimmune disease, metabolic syndrome, neurodegeneration, oncogenic and psychiatric disorders. The homo sapien species have a higher intake of dietary fibre contributing to around 40 g/day and a diverse microbial gut flora with less of archaeal population density. The butyrate generated from dietary fibre produces an immunosuppressive state. Thus the symbiotic microflora with less of archaeal density induces a homo sapien species. This can be demonstrated by experimental induction of evolution. A high fibre high MCT diet as well as antibiotics derived from higher plants and fecal microbiota transfer from sapien species can inhibit the Neanderthal metabolonomics and phenotype and induce the evolution of homo sapiens. A low fibre high fat high protein diet as well as fecal microbiota transfer from the Neanderthal species can produce Neanderthal metabolonomics and phenotype inducing the evolution of homo neanderthalis. Transfer of colonic microflora predominantly archaea and modulation of endosymbiotic archaea by a paleo diet and antibiotics from higher plants can lead to interconversion of human species between homo neanderthalis and homo sapiens. The hologenome especially the microbial flora endosymbiotic/gut drives human and animal evolution and can be experimentally induced. Symbiotic microflora drives evolution. Every animal, every human species, different communities, different races and different caste have their signature endosymbiotic and gut microflora which can be transmitted vertically and horizontally. Thus symbiosis drives human and animal evolution.

Methods for Species Change - Colonic Flora Probiotic Administration from Homo Sapiens and Homo Neanderthalis Identified by Blood Cytochrome F420 Activity

Research work carried out by us over a period of years has shown patients have this disorders or condition show a significant improvement on the natural organic paleo probiotic when endogenous archaeal growth and digoxin synthesis is demonstrated in the patients. Populations are screened for endosymbiotic archaeal activity in the sera by analysis of cytochrome F420 activity. The population that is negative for cytochrome F420 activity is chosen for the collection of the specimen. The blood cytochrome F420 negative population was taken as homo sapien phenotype. The population was fed on a paleo diet of high dietary fibre, high medium chain triglyceride and pulse/legume protein. The normal fecal collection was done from a healthy normal genetically related individual chosen by the patient and the administration of the organic natural probiotic isolated from the genetically related individual was volitional and a patient decision. The permission of the Ethics committee of the Institute - Metabolic Disorders Research Centre, Trivandrum was obtained. The fresh fecal matter from healthy humans was collected. Around 100 g of the organic matter is used in the preparation of the product. 100 g of the organic matter is diluted with normal saline and centrifuged at 2500 rpm. The rough matter forms a deposit and the supernatant is collected. The supernatant is preserved by adding 25 g of trehalose which can preserve the probiotic bacteria. This supernatant with added trehalose is freeze-dried and packed in double gelatin capsules. This capsule can be administered orally. The population with homo sapien characteristics was given fecal colonic flora preparation from neanderthalic phenotypes in the manner described above. The neanderthalic phenotypes were cytochrome F420 positive in their blood. Thus interconversion of species was possible by administration of probiotic

from colonic flora of homo sapiens and homo neanderthalis identified by cytochrome F420 activity in blood.

Methods for Species Change - High Fibre Diet Versus Low Fibre Diet

High archaeal growth induces neanderthalisation of human species. Neanderthal metabolonomics leads to chronic diseases like metabolic syndrome X, diabetes mellitus, CAD, stroke, autism, autoimmune, neuropsychiatric, neurodegenerative, cancer and infections. The patient in whom endogenous archaea and digoxin synthesis is demonstrated is given high fibre, legume protein and high medium chain triglyceride ketogenic diet along with natural antibiotics Curcuma longa, Moringa pterygosperma and Emblica officinalis ketogenic diet to modulate the effects of archaea and digoxin. This helps to convert the Neanderthal phenotype to homo sapien phenotype. Research work carried out by us over a period of years has shown neanderthalised species with civilizational disease as mentioned above show a significant improvement on the following combination when endogenous archaeal growth and digoxin synthesis is inhibited by a high fibre ketogenic diet derived from: (1) Curcuma longa, (2) Emblica officinalis, (3) Powdered Moringa pterygosperma, (4) Whole coconut powder, (5) Powdered black gram and (6) Powdered dried ash gourd. The individual materials were frozen dried and powdered to get 100-200 micron size. Then they were mixed at a concentration of: (1) 10 g of Curcuma longa - A, (2) 10 g of Emblica officinalis - B, (3) 100 g of Whole coconut powder - C, (4) 100 g of dried Moringa pterysperma leaves - D, (5) 100 g of Powdered dried black gram - E, and (6) 100 g of Powdered dried ash gourd - F. Components A, B, C, D, E and F were mixed to form a packet of 420 g. They were then mixed thoroughly and made into 420 g packet. They were assessed before treatment was started by clinical examination and lab investigations. The duration of the treatment ranged from 6 months to 2 years. We found that in the case tried high

fibre, legume protein and high medium chain triglyceride ketogenic diet along with natural antibiotics Curcuma longa, Moringa pterygosperma and Emblica officinalis showed significant curative effects. None of the substance used or information used in combination as described above for the purpose described to use have been used before. The consumption of a high fibre diet resulted in conversion of the homo neanderthalis species to homo sapien species. The high fibre diet results in reduction of gut archaeal growth and decreased endosymbiotic archaeal growth. The gut butyrate production is increased and the gut blood barrier and blood brain barrier is strengthened. The homo sapien species when fed a low fibre high fat high protein non-vegetarian diet has increased density of gut archaeal microflora and endosymbiotic archaeal growth. The gut butyrate generation is reduced and the gut blood barrier and blood brain barrier is breached. This leads to increase in endosymbiotic archaea and the homo sapien species gets converted to homo neanderthalis species.

Method of Interconversion of Human Species by Administering Colonic Microflora from Cow Dung

Archaeal symbiosis results in neanderthalisation of human species and civilizational diseases like metabolic syndrome X with diabetes mellitus and vascular disease, autoimmune, neuropsychiatric, neurodegenerative, cancer and infections. This invention relates to a formulation which will act as a natural organic paleo probiotic from dung of the Indian cow, Bos primigenius for various diseases which will inhibit archaeal growth and convert homo neanderthalis to homo sapiens. This disease process leading onto psychiatric disorders, tumours, autoimmune disease, metabolic syndrome and degenerations can be reversed by altering the gut microflora and populating it with non-archaeal phenotypes. This can be done by oral or rectal administration of fecal microflora of the Indian cow, Bos primigenius. The cow chosen for the purpose was the Indian cow, Bos primigenius. The Indian cow fed an organic

diet of grass and hay was chosen for the purpose. The administration of the organic natural probiotic isolated from the genetically related individual was volitional and a patient decision. The permission of the Ethics committee of the Institute - Metabolic Disorders Research Centre, Trivandrum was obtained. The fresh fecal matter from healthy the Indian cow, Bos primigenius are collected. Around 100 g of the organic matter is used in the preparation of the product. 100 g of the organic matter is diluted with normal saline and centrifuged at 2500 rpm. The rough matter forms a deposit and the supernatant is collected. The supernatant is preserved by adding 25 g of trehalose which can preserve the probiotic bacteria. This supernatant with added trehalose is freeze-dried and packed in double gelatin capsules. This capsule can be administered orally or as a rectal enema. Thus feeding of the colonic microflora from cow dung resulted in conversion of the Neanderthal metabolonomics to homo sapien metabolonomics.

Method of Interconversion of Species - Antioxidant Antibiotics

Archaeal symbiosis leads to neanderthalisation of the species with increased incidence of metabolic syndrome X, diabetes mellitus, CAD, stroke, autism, autoimmune, neuropsychiatric, neurodegenerative, cancer and infections. The patient in whom endogenous archaea and digoxin synthesis is demonstrated is given natural antioxidant antibiotics derived from crude extracts Curcuma longa, Moringa pterygosperma, Emblica officinalis, Zingiber officinale, Allium sativum and Withania somnifera to modulate the effects of archaea and digoxin. This converts the Neanderthal phenotype to homo sapien phenotype. Research work carried out by us over a period of years has shown patients have this disorders or condition show a significant improvement on the following combination when endogenous archaeal growth and digoxin synthesis is demonstrated in the patients. (1) Curcuma longa, (2) Emblica officinalis, (3) Powdered Moringa pterygosperma,

(4) Powdered Zingiber officinale, (5) Powdered Allium sativum, (6) Powdered Withania somnifera root and leaves. The individual materials were frozen dried and powdered to get 100-200 micron size. Then they were mixed at a concentration of (1) 10 g of Curcuma longa - A, (2) 10 g of Emblica officinalis - B, (3) 10 g of Powdered Moringa pterygosperma - C, (4) 10 g of Powdered Zingiber officinale - D, (5) 10 g of Powdered Allium sativum - E, (6) 10 g of Powdered Withania somnifera root and leaves - F. Components A, B, C, D, E and F were mixed to form a packet of 60 g. They were then mixed thoroughly and made into 60 g packet. They were assessed before treatment was started by clinical examination and lab investigations. The duration of the treatment ranged from 6 months to 2 years. We found that in the case tried natural antioxidant antibiotics derived from crude extracts Curcuma longa, Moringa pterygosperma, Emblica officinalis, Zingiber officinale, Allium sativum and Withania somnifera showed significant curative effects. None of the substance used or information used in combination as described above for the purpose described to use have been used before.

Details of the Trial

Archaeal symbiosis leads to neanderthalisation of the homo sapien species. This can be described as symbiosis mediated evolution. The homo neoneanderthalis has an increase predilection to metabolic syndrome X, strokes, CAD, hyperlipidemia, diabetes mellitus, autoimmune, neuropsychiatric, neurodegenerative, cancer and are retroviral resistant. The homo neanderthalis has different personality and social characteristics with increased creative, gender equal, matriarchal, asexual and alternate sexual, spiritual, intuitive, surrealistic and community centered characteristics. Neanderthal metabolonomics is primarily mediated by archaeal metabolonomics and archaeal symbiosis. They have got cholesterol catabolism, the shikimic acid

pathway, more of anaerobic glycolysis, increase connective tissue synthesis, fructolysis, nucleic acid synthesis and mitochondrial dysfunction. Self administration of the natural organic paleo probiotic from human colonic flora and cow dung, antioxidant antibiotic and high fibre high MCT diet to neanderthalised phenotype with pathological phenotypes of the following disorders. (1) Primary generalized epilepsy, (2) Schizophrenia, (3) Parkinson's disease, (4) Multiple sclerosis, (5) Refractory CNS glioblastomas, (6) Neuronal aging and dementia of the Alzheimer's type, (7) Down's syndrome, (8) Acquired immunodeficiency syndrome, (9) Autism, (10) CAD, (11) Stroke, (12) Diabetes mellitus, and (13) Aging. The patients were assessed before treatment was started clinically and by all required laboratory investigations. The duration of treatment ranged from 6 months to 2 years. Their condition was assessed during treatment and after treatment clinically and using all necessary laboratory investigations. This produced a change in the homo neanderthalis phenotype to homo sapien phenotype.

The homo sapien species are resistant to metabolic syndrome X, strokes, CAD, hyperlipidemia, diabetes mellitus, autoimmune, neuropsychiatric, neurodegenerative, cancer and are retroviral susceptible. The homo sapien species is less creative, patriarchal, gender unequal, heterosexual, logical and individualistic. Homo sapien metabolonomics is primarily aerobic and mitochondrial. The species change is a gut microflora and endosymbiotic flora mediated change which can be termed as induced evolution. The feeding of the homo sapien phenotype with a low fibre high fat high protein non-vegetarian diet resulted in increased in archaeal density in the gut microflora and endosymbiotic archaeal growth in the blood as measured by cytochrome F420 activity and neanderthalisation of the homo sapien species. This makes the homo sapien species neanderthalised with a different phenotype, genotype, psychological type and retroviral resistant.

Patient Population Included in the Large Scale Trial of Neanderthalised Phenotype

These are typical examples of a large number of patients tried in each case. The number of patients included in the trial is as follows. The neanderthalised phenotypes were fed a high fibre, high MCT vegetarian diet, colonic microflora probiotic from blood cytochrome F420 negative homo sapien population, colonic microflora from the Indian cow dung Bos primigenus and antioxidant antibiotic for 6 months showed conversion to homo sapien phenotypes with low blood cytochrome F420 activity and statistically significant disease remission. The psychological characters changed from neanderthalic increased creative, gender equal, matriarchal, asexual and alternate sexual, spiritual, intuitive, surrealistic and community centred characteristics to homo sapien less creative, patriarchal, gender unequal, heterosexual, logical and individualistic. The metabolic phenotype changed from neanderthalic cholesterol catabolism, the shikimic acid pathway, more of anerobic glycolysis, increase connective tissue synthesis, fructolysis, nucleic acid synthesis and mitochondrial dysfunction phenotype to homo sapien mitochondrial phenotype.

1. Primary generalized epilepsy - 25 patients.
2. Schizophrenia - 25 patients.
3. Parkinson’s disease - 25 patients.
4. Multiple sclerosis - 25 patients.
5. Refractory CNS glioblastoma - 15 patients
6. Diabetes mellitus - 50 patients
7. Neuronal aging and dementia of the Alzheimer’s type - 25 patients
8. Down’s syndrome - 15 patients
9. Acquired immunodeficiency syndrome - 15 patients
10. Autism - 50 patients
11. CAD - 50 patients

12. Stroke - 50 patients
13. Lupus syndrome - 25 patients

Patient population included in the large-scale trial of homo sapien phenotype identified by lower or absent cytochrome F420 activity in blood. They were fed a low fibre, high fat, high protein, non-vegetarian diet for 6 months. This resulted in increase in endosymbiotic and colonic archaeal density and neanderthalisation of the homo sapien phenotype. The homo sapien phenotype given colonic microflora capsules from normal Neanderthal phenotypes with high cytochrome F420 activity also resulted in neanderthalisation of homo sapien phenotype. The psychological characteristics changed from homo sapien less creative, patriarchal, gender unequal, heterosexual, logical and individualistic to neanderthalic increased creative, gender equal, matriarchal, asexual and alternate sexual, spiritual, intuitive, surrealistic and community centered characteristics. The metabolic phenotype changed from homo sapien mitochondrial phenotype to neanderthalic cholesterol catabolism, the shikimic acid pathway, more of anaerobic glycolysis, increase connective tissue synthesis, fructolysis, nucleic acid synthesis and mitochondrial dysfunction phenotype.

Summary

A method to induce evolutionary changes in the human species by modulating archaeal symbiosis and interconverting homo sapien to homo neanderthalis and vice versa is described. This is done by a high fibre versus a low fibre diet, administration of antioxidant antibiotic and colonic microflora from human and cow dung. This is a methodology to modulate species interconversion from homo sapien to homo neanderthalis with its attendant changes in psychological, phenotypic and metabolonomic characteristics of the population. This can be called as a therapeutic archaeal symbiotic modulated human evolution.

Chapter 4

Human Space Colonization and Human Re-engineering - Human Neanderthalis Metabolonomics - Generation of New Organelle

Human beings can be re-engineered to live in an anaerobic environment. This is possible by membrane sodium potassium ATPase inhibition and sodium potassium ATPase mediated ATP synthesis and by anaerobic glycolysis. Low level of EMF and photons can also mediate ATP synthesis via the electron transport chain. This occurs by inducing high level of archaeal symbiosis. This can be achieved by feeding low fibre diet, fecal transplantation and archaeal infusions. The archaea growth is increased in the gut and undergoes endosymbiosis generating new organelle - fructosoid, neurotransminoid, steroidelle, porphyrinoid, transmutoid and glycosaminoglycoid. This changes the human species to survive in an anaerobic environment or in an environment of ammonia or hydrogen sulphide. Archaeal symbiosis produces neanderthalisation of human beings which can survive in extremophilic environments including outside planetary system. The human systems exist as a habitat for an ecology of symbiotic bacteria and viruses. The human genes are small microislands floating in an ocean of bacterial and viral genes. The question of human body survival in extremophilic conditions of other planets can be solved by considering the survival of the symbiotic bacteria and viruses of the human systems. The human systems exist for the thriving and growth of the symbiotic bacteria and viruses. The induction of colonic and endosymbiotic archaeal symbiosis will induce changes in the ecosystem of endosymbiotic archaea, gut archaea, human tissues and archaeal phages. Metabolic engineering can be done for the survival of this ecosystem with human zombies functioning as the habitat for extremophilic symbiotic archaea. This is the crux for the survival of human beings in outer planetary system and establishing human colonies in outer planetary systems. This holds the key for human survival as a species and a race.

The endosymbiotic actinidic archaea forms the basis of life and can be considered as the third element in the cell. It regulates the cell, the neuro-immune-endocrine system and the conscious / unconscious brain. The

endosymbiotic actinidic archaea can be called as the elixir of life. A definite population of endosymbiotic actinidic archaea is required for the existence and survival of life. A higher density of endosymbiotic actinidic archaeal population can lead to human disease. Thus actinidic archaea are important for survival of human life and can be considered as crucial to it. The endosymbiotic actinidic archaea forms the basis of life and can be considered as the third element in the cell. It regulates the cell, the neuro-immune-endocrine system and the conscious/unconscious brain. The endosymbiotic actinidic archaea can be called as the elixir of life. A definite population of endosymbiotic actinidic archaea is required for the existence and survival of life. A higher density of endosymbiotic actinidic archaeal population can lead to human disease. Thus actinidic archaea are important for survival of human life and can be considered as crucial to it. Symbiosis by actinidic archaea is the basis of evolution of humans and primates. The increase in endosymbiotic archaeal growth can lead to the induction of homo neanderthalis. This endosymbiotic archaea induced neanderthalisation of the species leads to human disease like metabolic syndrome X, neurodegenerations, schizophrenia and autism, autoimmune disease and cancer. The reduction in endosymbiotic archaeal growth by a high fibre, high medium chain triglyceride and legume protein ketogenic diet, antibiotics from higher plants like Curcuma longa, Emblica officianalis, Allium sativum, Withania somnifera, Moringa pterygosperma and Zingeber officianalis and transplantation of colonic microflora from normal homo sapien population can lead to deneanderthalisation of species and treatment of the above mentioned diseased states. The colonic microflora of neanderthalised diseased states like metabolic syndrome X, neurodegenerations, schizophrenia and autism, autoimmune disease and cancer when transferred to the normal homo sapien species leads to generation and induction of homo neanderthalis. Thus primate and human evolution is symbiotic event which can be induced the

modulating symbiotic archaeal growth. Human populations can be divided into matrilineal Neanderthal population in South Indian Dravidians, Celts, Basques, Jews and Berbers and the Cro-Magnon population seen in Africa and Europe. The symbiotic archaeal colonization decides which species - Neanderthal or Cro-Magnon to which the society belongs to. It is tempting to postulate symbiotic microflora and archaea determining the family behavior and traits as well as societal and caste behavior and traits. The cell has been postulated by Margulis to be a symbiotic association of bacteria and viruses. Similarly, the family, the caste, the community, nationalities and the species itself is determined by archaeal and other bacterial symbiosis. Symbiosis by microorganisms especially archaea drives the evolution of the species. In such a case symbiosis can be induced by transfer of microflora symbionts and evolution induced. Transfer of colonic microflora predominantly archaea and modulation of endosymbiotic archaea by a paleo diet and antibiotics from higher plants can lead to interconversion of human species between homo neanderthalis and homo sapiens.

Symbiosis by microorganisms especially archaea drives the evolution of the species. In such a case symbiosis can be induced by transfer of microflora symbionts and evolution induced. Endosymbiosis by archaea as well as archaeal symbionts in the gut can modulate the genotype, the phenotype, the social class and the racial group of the individual. The symbiotic archaea can have horizontal and vertical transmission. Endosymbiotic archaeal growth leads to neanderthalisation of the species. The neanderthalised species is matrilineal society and includes the Dravidians, the Celts, the Basques and the Berbers. The inhibition of the endosymbiotic archaeal growth leads to evolution of the homo sapiens. This includes the Africans, Aryan invaders of North India and the Aryan derived European population. Symbiosis mediated evolution depends on the gut flora and the diet. This has been demonstrated in the drosophila pseudoobscura. The drosophila mates only with other individuals eating the

same diet. When the drosophila gut microflora is altered by feeding antibiotics they mate with other individuals eating different diets. The diet consumed by the drosophila regulates its gut microflora and mating habits. The combination of the human genome and the symbiotic microbial genome is called the hologenome. The hologenome especially its symbiotic microbial component drives human evolution as well as animal evolution. The evolutionary distance between species of wasp depends on the gut microflora. The human gut microflora regulates the endocrine, genetic and neuronal systems. Humans and primate evolution depends on endosymbiotic archaea and gut microflora. The endosymbiotic archaeal growth determines the racial differences between the matrilineal Harappan/Dravidian societies and the patriarchal Aryan society. The matrilineal Harappan / Dravidian society was neanderthalic and had increased endosymbiotic archaeal growth. Endosymbiotic archaeal growth and neanderthalisation can lead to autoimmune disease, metabolic syndrome X, neurodegeneration, cancer, autism and schizophrenia. The Neanderthal gut flora and endosymbiotic archaea was determined by the non vegetarian ketogenic high fat high protein diet consumed by them in the Eurasian steppes. The homo sapiens including the classical Aryan tribes and African ate a high fibre diet and had lower archaeal growth both endosymbiotic and gut. The dietary fibre intake determines the microbial diversity of the gut. The high fibre intake is associated with increased generation of short chain fatty acids - butyric acid by the gut flora. Butyrate is a HDAC inhibitor and leads to increased generation and incorporation of endogenous retroviral sequences. The high dietary fibre intake related increased HERV sequences leads to increased synaptic connectivity and a dominant frontal cortex as seen in homo sapien species. The neanderthalic species consume a ketogenic non-vegetarian high fat high protein low fibre diet. This leads to decreased generation of endogenous HERV sequences and reduced genomic flexibility in neanderthalic species. This produces smaller

cerebral cortex and a dominant cerebellar cortex in the neanderthalic brain. The homo neanderthalic species by the low dietary fibre intake starve their microbial self. This leads to increased endosymbiotic and gut archaeal growth. The mucous membrane lining the gut becomes thinned out as the gut bacteria eats up the mucous lining of the gut. This results in leakage of endotoxin and archaea from the gut to the blood breaching the barrier and produces a chronic immunostimulatory inflammatory state which forms the basis of autoimmune disease, metabolic syndrome, neurodegeneration, oncogenic and psychiatric disorders. The Neanderthal species eat a low fibre diet and have a deficiency of microbiota accessed carbohydrate generating short chain fatty acid. There is a deficiency of butyrate generated in the gut from the dietary fibre which can produce suppression of the chronic inflammatory process. The Neanderthals have got the fermentation by-product deficiency syndrome. The induction of neanderthalic species depends on the low fibre intake induced high archaeal density endosymbiotic and the gut microflora. The homo sapiens species consume a high fibre diet generating large amounts of short chain fatty acid butyrate which inhibits endosymbiotic and gut archaeal growth. The microbial self of the homo sapien species is more diverse than that of the neanderthalic species and the archaeal population density is less. This results in a protection against chronic inflammation and the induction of diseases like autoimmune disease, metabolic syndrome, neurodegeneration, oncogenic and psychiatric disorders. The homo sapien species have a higher intake of dietary fibre contributing to around 40 g/day and a diverse microbial gut flora with less of archaeal population density. The butyrate generated from dietary fibre produces an immunosuppressive state. Thus the symbiotic microflora with less of archaeal density induces a homo sapien species. This can be demonstrated by experimental induction of evolution. A high fibre high MCT diet as well as antibiotics derived from higher plants and fecal microbiota transfer from sapien

species can inhibit the Neanderthal metabolonomics and phenotype and induce the evolution of homo sapiens. A low fibre high fat high protein diet as well as fecal microbiota transfer from the Neanderthal species can produce Neanderthal metabolonomics and phenotype inducing the evolution of homo neanderthalis. Transfer of colonic microflora predominantly archaea and modulation of endosymbiotic archaea by a paleo diet and antibiotics from higher plants can lead to interconversion of human species between homo neanderthalis and homo sapiens. The hologenome especially the microbial flora endosymbiotic/gut drives human and animal evolution and can be experimentally induced. Symbiotic microflora drives evolution. Every animal, every human species, different communities, different races and different caste have their signature endosymbiotic and gut microflora which can be transmitted vertically and horizontally. Thus symbiosis drives human and animal evolution. The colonic and endosymbiotic archaea and other microbes like clostridial clusters determine the species, race, caste, community and personal identity of the individual. The identity of the individual - personal, community, caste, race, nationality and species is determined by the colonic and endosymbiotic archaeal and clostridial clusters. Predominant archaeal symbiosis produces homo neanderthalis and less prominent archaeal symbiosis and dominant clostridial clusters in the gut produces the homo sapien species. Each individual, race, nationality, caste, creed and community have the endosymbiotic and colonic microbiota signature. This colonic and endosymbiotic microbiota signature is transferable by the change of endosymbiotic and colonic microbiota from one group to another. Thus the evolution and identity based on individuality, race, nationality, caste and creed can be induced.

This can be interpreted on the basis of Villarreal hypothesis of group identity and cooperativity of RNA collectives. Archaeal symbiosis in the gut and in the tissue spaces determines speciation of human beings as homo sapiens and homo

neanderthalis. The endosymbiotic archaea can secrete RNA viroids and viruses and there is a viroid-archaeal host relationship between the two. A dynamic state of virus lysis and persistence can occur in archaea suggesting that viral addiction can occur in archaea. The RNA viroids in the archaea coordinate their behavior by information exchange, modulation and innovation generating new sequence based content. This occurs due to a phenomenon of symbiosis in contrast to the concept of survival of the fittest. The generation of new RNA viroidal sequences is a result of practical competence of living agents to generate new sequences by symbiosis and sharing. This represents highly productive RNA viroidal quasi-species consortia for the evolution, conservation and plasticity of genomic environments. The behavioural motives of the RNA are single stem loop structures. They have self folding and group building capabilities depending upon functional needs. The evolution process depends upon what Villareal calls RNA stem loop consortia. The whole entity can function only if participatory groups of RNA viroids can get their function coordinated. There is competent denovo generation of new sequences by cooperative action and not by competition. These RNA viroidal group consortia can contribute to the host identity, group identity and group immunity. The term used for this is RNA viroidal sociological behavior. The RNA viroids can build groups that invade the archaea and compete as a group for limited resources such host genomes. A key behavioural motif is able to integrate a persistent life style into the archaeal colony with the addiction module forming competing viroidal groups that are counter balancing each other together with the archaeal/host immune system. This leads to creation of an identity for the archaeal colony and the homo neanderthalis host. Viroids can kill their host and also colonize their host without disease and protect the host from similar viruses and viroids. Together with lysis and protection we see a viroid colonized host that is both symbiotic and innovative acquiring new competent codes. Thus the

viroid-host relationship is a pervasive, ancient force in the origin and evolution of life. Cumulative evolution at the level of RNA viroids is like a ratchet effect used for transmission of cultural memes. This learning accumulates so that every new generation must not repeat all innovative thoughts and techniques. Quasi-species of RNA viroids are cooperative and exclusive of other quasi-species. They have group recognition differentiating self-groups and non-self-groups allowing for quasi-species to promote the emergence of group identity. With group identity via counter related addiction modules two opposing components must be present and work coherently and define the group as a whole. Biological identity is constituted by dynamic interaction of cooperative groups. Virus addiction module is an essential strategy for existence of life in the virosphere. Viruses are transmissible and can persist in specific host population leading to a form of group immunity / identity since identical but uncolonized host population remains susceptible to a killing action of lytic viruses. In this way we see that viruses are necessary providing opposing functions for addiction (persistence/protection and lytic/killing). Viroids can function as consortia, an essential interacting group and provide a mechanism from which consortial function could emerge in the origin of protobiotic life. Genetic parasites can act as a group (qs-c). But for this group to be coherent they must attain group identity and this is typically via an addiction strategy. Antiviral and proviral system in the archaea will themselves emerge in the host from virus derived information. The archaeal viruses themselves provide the critical function required for antiviral defence. The opposing functions are the basis of addiction modules. Thus the emergence of group identity becomes an essential and early event in the emergence of life. This is coherent to the basically group behavior of RNA viroids in archaea. This group selection and group identity are needed to create information coherence and network formation and to establish a system of communication - code competent

interactions. This identity serves as information also for the ones that do not share this identity. This is the beginning of self/non-self differentiating capability. In this way viroids promote the emergence of group identity in archaeal colonies and host humans. The archaeal colony identity depends upon the colonizing set of RNA viroids producing a coherent network that is inclusive opposing functions and favours the persistence of parasite derived new information. On the basis of population-based functions of RNA DNA can be considered as a habitat for consortia RNA. Thus RNA viroids of the archaea are involved in complex multicellular identity. This is called as the Gangen hypothesis by Villarreal. The Gangen describes the emergence of commonly shared code use, group membership and collective living function of RNA viroids. Communication is a code depended interaction and transmission of infectious code defines the origin of the virosphere. This issue refers to the idea of collective of RNA viroids with inherent toxic and antitoxic features should be able to transmit or communicate these agents and their features to a nearby competing population. It strongly favours the survival of RNA viroidal population with compatible addiction modules that will inhibit agent toxicity and allow persistence of new agents. This is thus the survival of the persistently colonized set which is an inherently symbiotic and consortial process. It also promotes increasing complexity and identity/immunity of the host collective via a new agent colonization, and stable addition. Thus the transmission of RNA agents attains both communication and recognition of group membership. In this way the emergence of the virosphere must had been an early event in the origin of life and group identity. Viruses and viroids are genetic parasites and the most abundant living entities on earth. The virosphere is a network of infectious genetic agents. Evolution, conservation and plasticity of genetic identities are the result of cooperative consortia of RNA viroids that are competent to communicate. Thus the archaeal viroidal consortia can

symbiotically share and communicate producing new sequences and give an identity to the archaeal colony. The low fibre diet and extreme temperatures of the Eurasian steppes leads to archaeal multiplication and induction of the homo neanderthalis species. The archaeal colony's characteristics are determined by the cooperative consortia of RNA viroids in the archaea and the archaeal colony identity determines the homo neanderthalis identity. Thus the archaeal colonies with their quasi-species consortia of RNA viroids determine the homo neanderthalis identity. The new sequence generation by the RNA viroidal consortia's symbiotic sharing character contributes to the diversity in the behavior and creativity of the homo neanderthalis population. The archaeal RNA viruses and viroids and the archaeal colonies themselves protect the homo neanderthalis population from retroviral infections. Thus the homo neanderthalis population is retroviral resistant and the quasi-species consortia of archaea and archaeal viroids gives them a group identity as retroviral resistant. Thus the quasi-species consortia of archaea and RNA viroids give homo neanderthalis colonies their identity and idea of self. The homo neanderthalis is resistant to retroviral infection like the Australian aboriginals and the endogenous retroviral sequences in the Neanderthal genome are limited. This leads to lack of plasticity and dynamicity of the human genome and the cerebral cortex in ill-developed with a dominant impulsive cerebellar cortex in the homo neanderthalis population. This produces the impulsive creative surrealistic spiritual neanderthalic brain. As the extreme of temperature goes off and the ice age ends the archaeal population density also comes down. This also can result from the consumption of a high fibre diet in the African continent. The high fibre diet digested by clostridial clusters in the colon promotes butyrate synthesis and butyrate will induce HDAC inhibition and expression of retroviral sequences in the primate genome. This leads to increase in endogenous retroviral sequences in the human genome, increasing genomic dynamicity and

the evolution of complicated cerebral cortex dominant brain with its complex synaptic connectivity in the homo sapiens. This leads onto a logical, commonsensical, pragmatic and practical homo sapien brain. The homo sapiens due to lack of archaea and the RNA viroids are susceptible retroviral infection. Thus the archaeal colonies and RNA viroidal quasi-species consortia determine the evolution of the human species and the brain networks. Thus extremes of temperature, fibre intake, archaeal colony density, RNA viroidal quasi-species, group identity and retroviral resistance decides on the evolution of homo sapiens and homo neanderthalis as well as the brain networks. The present extremes of temperature and low fibre intake in civilized society can lead to increase in archaeal population densities and quasi-species RNA viroidal networks generating a new homo neanderthalis in a new neanderthalic anthropocene age as opposed to the present homo sapien anthropocene age. The archaeal population densities and quasi-species RNA viroidal networks determine homo sapien / homo neanderthalis species, racial, caste, community, national, sexual, metabolic, phenotypic, neuronal, psychiatric, psychological, immune, genotypic and individual identity. The archaea secretes the trephone digoxin which can edit the RNA viroids and generate new sequences. Archaeal dipolar magnetite and porphyrins in the setting of digoxin induced membrane sodium potassium ATPase inhibition can produce a pumped phonon system mediated quantal perceptive state and quantal communication in the RNA viroidal symbiotic system generating new sequences by steroidal digoxin enzymatic editing action. This gives rise to archaeal RNA viroidal quasi-species symbiotic diversity and identity to species, race, caste, sex, culture, individual and national identity.

The roots of Western civilizational disease can be related to the starvation of the colonic microflora. The colonic microflora depends upon complex carbohydrates derived from dietary fibre. The processed food of high protein,

fat and sugars is digested and absorbed in the stomach and small intestine. A very little of it reaches the colon and widespread use of antibiotics in medicine has produced mass extinction of the colonic microflora. The colonic microflora is extremely diverse and the diversity is lost. There are 100 trillion bacteria in the colon belonging to 1200 species. They regulate the immune system by inducing the T-regulatory cells. A high fibre diet contributes to colonic microbiota diversity. Interaction with farm animals like cows and dogs also contributes to the colonic microflora diversity. The typical Western diet of high fat, high protein and sugars decreases the colonic microbiota diversity and increase colonic/endosymbiotic archaea producing methanogenesis. The colonic archaea feed upon the mucous lining of the colon and produces leakage of archaea into the blood and tissue system producing endosymbiotic archaea. This results in a chronic inflammatory state. The high fibre diet of Africans, South Americans and Indians produces increased colonic microbiota diversity and increase in clostridial clusters generating SCFA in the gut. High fibre diet is protective against metabolic syndrome and diabetes mellitus. Metabolic syndrome is related to degeneration, cancer, neuropsychiatric illness and autoimmune disease. A high fibre diet of upto 40 g/day can be called as a gut diet. The colonic microflora especially the clostridial cluster digests the fibre generating short chain fatty acids which regulates immunity and metabolism. High fibre diet increases the colonic mucus secretion and the thickness of the mucus lining. A high fibre diet produces increase in clostridial clusters and mucous secretion. This produces a strong gut blood barrier and prevents metabolic endotoxemia which produces a chronic inflammatory response. High dietary fibre intake and the diversity of the colonic microflora with prominent SCFA producing clostridial clusters are interrelated. The clostridial clusters metabolise the complex carbohydrate in dietary fibre to short chain fatty acids butyrate, propionate and acetate. They increase the T-regulatory function. A

high fibre diet increases the bacteroides and reduces the firmecutes of the colonic microflora. A high fibre diet is associated with a low body-mass index. A low fibre diet produces increase in colonic archaeal growth as well as endosymbiotic tissue and blood archaea. This produces more of methanogenesis rather than short chain fatty acid synthesis contributing to immune activation. A low fibre diet is associated a high body-mass index and chronic systemic inflammation. Germ-free mice show cardiac, pulmonary and liver atrophy. Gut microflora is required for the generation of organ systems. The gut microflora is also required for generation of T-regulatory cells. High fibre intake produces more colonic microbiota diversity and increase in clostridial clusters and fermentation by products like butyrate which suppresses inflammation and increases T-regulatory cells. A low fibre diet produces increase in archaeal growth, methanogenesis, destruction of the mucus lining and leakage of the colonic archaea producing endosymbiotic tissue and blood archaea. This produces an immune hyperreactivity contributing to the modern plagues of civilization - metabolic syndrome, schizophrenia, autism, cancer, autoimmunity and degenerations. The gut microbiota drives human evolution. The humans don't host the gut microbiota but the gut microbiota host us. The human system forms an elaborate culture laboratory for the propagation and survival of the microbiota. The human system is induced by the microbiota for their survival and growth. The human system exists for the microbiota and not the other way round. The same mechanism holds good in plant systems. Plant started the colonized earth as they started symbiosing with bacteria in the roots systems which can derive nutrients from the soil. Human beings form a mobile culture laboratory for the more effective propagation and survival of the microbiota. The microbiota induces the formation of specialized immune cells called innate lymphoid cells. The innate lymphoid cells will direct the lymphocytes not to attack the beneficial bacteria. Thus the endosymbiotic archaea and the gut

archaea induce human, primate and animal evolution to generate structures for them to survive and propagate. The source of endosymbiotic archaea, the third element of life is the colonic archaea that leaks into the tissue spaces and blood systems due to breach in the gut blood barrier. The increase in colonic archaea is due to the starvation of the gut microbiota consequent to a low fibre diet. This results in increase in colonic archaeal growth and destruction of clostridial clusters and bacteroides. The increase colonic archaeal growth in the presence of gut starvation due to low fibre diet eats up the mucus lining and produces breakages in the gut blood barrier. The colonic archaea enters the blood stream and produces endosymbiosis generating endosymbiotic archaea and various new organelle - fructosoids, steroidelle, vitaminocyte, viroidelle, neurotransminoid, porphyrinoids and glycosaminoglycoids.

The human brain can be considered as a modified archaeaon colony network. The archaeaon are eternal and can last for billions of years. The human brain is basically an information storage system. The archaeaon has got dipolar magnetite and porphyrins and can function as quantal computer. The archaeal colony with its dipolar magnetite and porphyrin in the setting of archaeal digoxin induced membrane sodium potassium ATPase inhibition can function as a pumped phonon system mediating quantal perception. The archaeaon in the brain is capable of information storage at a point in time and space. The experiences and information stored in the archaeaon is immortal and eternal. The archaeaon can have a wave particle existence and can exist in multiple quantal possible states and can inhabit multiple quantal multiverses. The interaction between information stored in quantal computers in multiple different archaeaon systems all over the universe by the quantal interactions results in eternal existence of information in quantal multiverses. The information in the quantal multiverses can have a particulate existence creating a newer mode by quantal interactions between information stored at multiple

points of time. This creates the particulate mythic world of human existence. These are what are called as Samsaras. The mind is uploaded into information in the neuronal archaeal colony network and its quantal computers. The information stored in the archaeal colony network mediated quantal state is eternal and can be considered as a digital version of the brain, a mind downloading technique or whole brain emulation. The archaeal colony network stores the human experiences in an eternal manner and can contribute to biological reincarnation.

Global warming induces endosymbiotic archaeal and RNA viroidal growth. The porphyrins form a template for the formation of RNA viroids, DNA viroids, prions, isoprenoids and polysaccharides. They can symbiose together to form primitive archaea. The archaea can further induce HIF alpha, aldose reductose and fructolysis resulting in further porphyrinogenesis and archaeal self replication. The primitive archaeal DNA is integrated along with RNA viroids which are converted to their corresponding DNA by the action of redox stress induced HERV reverse transcriptase into the human genome by the redox stress induced HERV integrase. The archaeal DNA sequences that are integrated into the human genome forms endogenous archaeal human genomic sequences akin to HERV sequences and can function as jumping genes regulating genomic DNA flexibility. The integrated endogenous genomic archaeal sequences can get expressed in the presence of redox stress forming endosymbiotic archaeal particles which can function as a new organelle called the archaeaons. The archaeaon can express the fructolytic pathway constituting an organelle called the fructosome, cholesterol catabolic pathway and digoxin synthetic forming an organelle called the steroidelle, the shikimic acid pathway forming an organelle called the neurotransminoid, antioxidant vitamin E and vitamin C synthetic organelle called the vitaminocyte as well as the glycosaminoglycan synthetic organelle called glycosaminoglycoid. The archaea can secrete capsulated RNA

viroidal particles which can function as blocking RNAs modulating cell metabolism and such archaeaon organelle are called viroidelle. The archaea suppresses pyruvate dehydrogenase and promotes fructolysis resulting in accumulation of pyruvate which enters the GABA shunt pathway producing succinyl CoA and glycine, the substrates for porphyrin synthesis. Porphyrin forms a template for the formation of RNA viroids, DNA viroids, prions and isoprenoids which can symbiose together to form an archaea. Thus endosymbiotic archaea have an abiogenic replication. The archaeaon concerned with GABA shunt pathway and porphyrinogenesis are called porphyrinoids. The archaeaon colony forms a network with different areas showing differential specialization of function - fructosoids, steroidelle, vitaminocyte, viroidelle, neurotransminoid, porphyrinoids and glycosaminoglycoids. This forms a living organized structure within human cells and tissues regulating their function and reducing the human body to zombie working under the directions of the organized archaeal colony. The organized archaeal colony has abiogenetic replication and is eternal.

The increase in endogenous EDLF, a potent inhibitor of membrane Na^+-K^+ ATPase, can decrease this enzyme activity. The results showed increased endogenous EDLF synthesis as evidenced by increased HMG CoA reductase activity, which functions as the rate limiting step of the isoprenoid pathway. Studies in our laboratory have demonstrated that EDLF is synthesized by the isoprenoid pathway. The endosymbiotic archaeal sequences in the human genome get expressed by redox stress and osmotic stress of global warming. This results in induction of HIF alpha which will upregulate fructolysis and glycolysis. In the setting of redox stress all glucose gets converted to fructose by the induction of enzymes aldose reductase and sorbitol dehydrogenase. Aldose reductase converts glucose to sorbitol and sorbitol dehydrogenase converts sorbitol to fructose. Since fructose is preferentially phosphorylated by ketohexokinases the cell is depleted of ATP and glucose phosphorylation comes

to a halt. Fructose becomes the dominant sugar that is metabolized by fructolysis in expressed archaeal particles in the cell functioning as organelle called fructosoids. The fructose is phosphorylated to fructose 1-phosphate which is acted upon by aldolase B which converts it into glyceraldehyde 3-phosphate and dihydroxy acetone phosphate. Glyceraldehyde 3-phosphate is converted to D 1,3-biphosphoglycerate which is then converted to 3-phosphoglycerate. The 3-phosphoglycerate is converted to 2-phosphoglycerate. 2-phosphoglycerate is converted to phosphoenol pyruvate by the enzyme enolase. Phosphoenol pyruvate is converted to pyruvate by the enzyme pyruvic kinase. The archaeaon induces HIF alpha which upregulates fructolysis and glycolysis but inhibits pyruvate dehydrogenase. The forward metabolism of pyruvate is stopped. The dephosphorylation of phosphoenol pyruvate is inhibited in the setting of pyruvic kinase inhibition. Phosphoenol pyruvate enters the shikimic acid pathway where it is converted to chorismate. The shikimic acid is synthesized by a pathway starting from glyceraldehyde 3-phosphate. Glyceraldehyde 3-phosphate combines with the pentose phosphate pathway metabolite sedoheptulose 7-phosphate which is converted to erythrose 4-phosphate. The pentose phosphate pathway is upregulated in the presence of the suppression of glycolytic pathway. Erythrose 4-phosphate combines with phosphoenol pyruvate to generate shikimic acid. Shikimic acid combines with another molecule of phosphoenol pyruvate to generate chorismate. The chorismate is converted to prephenic acid and then to parahydroxy phenyl pyruvic acid. Parahydroxy phenyl pyruvic acid is converted to tyrosine and tryptophan as well as neuroactive alkaloids. The shikimic acid pathway is structured in expressed archaeaon organelle called the neurotransminoid. The fructolytic intermediates glyceraldehydes 3-phosphate and pyruvate are the starting points of the DXP pathway of cholesterol synthesis. Glyceraldehyde 3-phosphate combines with pyruvate to form 1-deoxy D-xylulose phosphate

(DOXP) which is then converted to 2-C methyl erythritol phosphate. 2-C methyl erythritol phosphate can be synthesized from erythrose 4-phosphate a metabolite of the shikimic acid pathway. DXP combines with MEP to form isopentenyl pyrophosphate which is converted to cholesterol. Cholesterol is catabolized by archaeal cholesterol oxidases to generate digoxin. The digoxin sugars digitoxose and rhamnose are synthesized by the upregulated pentose phosphate pathway. Glycolytic suppression leads to upregulation of the pentose phosphate pathway. The expressed archaeaon organelle concerned with cholesterol catabolism and digoxin synthesis is called the steroidelle. The suppression of glycolysis and stimulation of fructolysis results in upregulation of the hexosamine pathway. Fructose is converted to fructose 6-phosphate by ketohexokinases. The fructose 6-phosphate is converted to glucosamine 6-phosphate by the action of glutamine fructose 6-phosphate amidotransferase (GFAT). Glucosamine 6-phosphate is converted to UDP N-acetyl glucosamine which is then converted to N-acetyl glucosamine and various amino sugars. UDP glucose is converted to UDP D-glucuronic acid. UDP D-glucuronic acid is converted to glucuronic acid. This forms the uronic acid synthetic pathway. Uronic acids and hexosamines form repeating units of glycosaminoglycans. In the setting of glycolytic suppression and fructolytic metabolism fructolysis leads to increase synthesis of hexosamines and GAG synthesis. The GAG synthesizing archaeaon particles are called the glycosaminoglycoids. The expressed archaeaon particles are capable of synthesizing antioxidant vitamin C and E. The UDP D-glucose is converted to UDP D-glucuronic acid. UDP D-glucuronic acid is converted to D-glucuronic acid. D-glucuronic acid is converted to L-gulonate by enzyme aldoketoreductases. L-gulonate is converted to L-gulonolactone by lactonase. L-gulonolactone is converted to ascorbic acid by the action of archaeal L-gulo oxidase. The vitamin E is synthesized from shikimate which is converted to tyrosine and then to parahydroxy phenyl

pyruvic acid. Parahydroxy phenyl pyruvic acid is converted to homogentisate. Homogentisate is converted to 2-methyl 6-phytyl benzoquinone which is converted to alpha tocopherol. 2-methyl 6-phytyl benzoquinone is converted to 2,3-methyl 6-phytyl benzoquinone and gamma tocopherol. Vitamin E can also be synthesized by the DXP pathway. Glyceraldehyde 3-phosphate and pyruvate combined to form 1-deoxy D-xylulose 5-phosphate which is converted to 3-isopentenyl pyrophosphate. 3-isopentenyl pyrophosphate and dimethyl allyl pyrophosphate combined to form 2-methyl 6-phytyl benzoquinone which is converted to tocopherols. The ubiquinone another important membrane antioxidant and part of the mitochondrial electron transport chain is synthesized by the shikimic acid pathway and DXP pathway. The isoprenoid moiety of ubiquinone is contributed from the DXP pathway and the rest of it by tyrosine catabolism. The tyrosine is generated by the shikimic acid pathway. The archaeaon particles concerned with the synthesis of vitamin C, vitamin E and ubiquinone which are all antioxidants are called the vitaminocyte.

Global warming induces endosymbiotic archaeal and RNA viroidal growth. The endosymbiotic archaea and the generated RNA viroids induce aldose reductase which converts glucose to sorbitol. The archaeal polysaccharides and lipopolysaccharides as well as viroids and viruses can induce aldose reductase. Sorbitol is acted upon by sorbitol dehydrogenase to generate fructose which enters fructolytic pathway. Aldose reductase is also induced by the osmotic stress of global warming and redox stress. Aldose reductase is induced by inflammatory and immune stimulation. Archaeal synthesized endogenous digoxin can produce intracellular redox stress and activate NFKB which produces immune activation. Both redox stress and immune activation can activate aldose reductase which converts glucose to fructose. Hypoxic stress or anaerobic conditions induces HIF alpha which activates ketohexokinase C which phosphorylates fructose. Fructose is acted upon by fructokinase which

converts fructose to fructose 1-phosphate. Fructose 1-phosphate is converted to dihydroxy acetone phosphate and glyceraldehydes 3-phosphate which is converted to pyruvate, acetyl CoA and citrate. Citrate is used for lipid synthesis. Fat deposition occurs in the visceral organs like the liver, heart and kidney. There is no subcutaneous fat deposit. Fructose metabolism bypasses phosphofructokinase which is inhibited by citrate and ATP. Fructose metabolism is therefore not under the regulatory control of the enzyme phosphofructokinase. Fructose transport and metabolism is not regulated by insulin. Fructose is transported by glut 5 receptor. Fructose does not increase insulin secretion and therefore does not activate lipoprotein lipase. This results in visceral adipogeneis. Fructose induces ChREBP and SREBP elements. This results in increased hepatic lipogenesis by the induction of the enzyme fatty acid synthase, acetyl CoA carboxylase and steroyl CoA desaturace. This increases fatty acids and cholesterol synthesis. Fructose is a lipophilic carbohydrate. Fructose can be converted to glycerol 3-phosphate and fatty acids involved in triglyceride synthesis. Fructose administration leads to increase in triglycerides and VLDL. Fructose consumption leads to insulin resistance, fat accumulation in visceral organs like liver, heart and kidney, insulin resistance, dyslipidemia with increased triglycerides, VLDL and LDL as well as the metabolic syndrome. The metabolic syndrome x can be considered as a fructolytic syndrome. Fructose will increase lipid storage and promote insulin resistance. Fructose can fructosylate proteins producing dysfunction. Fructose has no effect upon ghrelin and leptin in the brain and can lead to increased feeding behaviour. Glucose decreases ghrelin and increases leptin levels. This leads to suppression of appetite. Thus fructose can modulate eating behaviour leading onto obesity. Fructose results in NFKB activation and TNF alpha secretion. TNF alpha can modulate the insulin receptor producing insulin resistance and metabolic syndrome x. Fructose

can also lead to leptin resistance and obesity. There is an epidemic of metabolic syndrome x in relation to global warming.

Fructose can activate the sympathetic nervous system. This leads to hypertension and increase in heart rate. Fructose is involved in left ventricular hypertrophy, increase in left ventricular mass and decrease in left ventricular ejection fraction in hypertension. Fructose suppresses the parasympathetic nervous system. Fructose acts as a key inducer for uncontrolled proliferation and hypertrophy of the cardiac musculature consequent to hypertension. The heart uses beta oxidation of fatty acids to generate energy. In the setting of anerobic glycolysis consequent to myocardial infarction and hypertensive hypertrophy of the heart, there is induction of HIF alpha. This produces increase in ketohexokinase C in the heart which phosphorylates fructose. Ketohexokinase C is a predominant liver enzyme as fructose metabolism is primarily focused in the liver. In the setting of anaerobic glycolysis ketohexokinase C is also produced in the brain and the heart. Ketohexokinase A is the predominant enzyme in the heart and brain. In the setting of anaerobic glycolysis ketohexokinase A which preferentially metabolizes glucose is converted to ketohexokinase C metabolizing fructose by the mechanism of RNA splicing. Anerobic conditions can induce HIF alpha which activates the splicing factor SF3B1. Thus HIF alpha induced by glycolysis induces SF3B1 which induces ketohexokinase C producing fructolysis in the heart. The fructose is converted to lipids, glycogen and glycosaminoglycans in the heart producing cardiac hypertrophy. Fructose metabolism is not under regulatory control of the key enzyme phosphofructokinase by citrate and ATP. The fructolytic pathway functions as a rogue pathway not under any regulatory control. Fructose is a key contributor. The sympathetic overactivity and parasympathetic blockade consequent to fructose can produce immune activation. The sympathetic

overactivity and parasympathetic blockade can lead to dysregulation of the nervous system.

Fructose can activate NFKB and tumour necrosis factor alpha. The vagal blockade produced by fructose also leads to increase in immune activation. Fructose can inhibit neutrophilic phagocytosis. Increased fructose ingestion can lead to immune activation and respiratory diseases like chronic bronchitis, COPD and bronchial asthma as well as interstitial lung disease. This immune activation induced by fructose is called as fructositis. Fructosylated proteins can serve as autoantigens. Fructosylated proteins can bind to RAGE receptors producing immune activation. Global warming induced fructose disease is the basis of the epidemic of autoimmune disease rising with the global warming.

Fructose increases flux through the pentose phosphate pathway. This increases the availability of hexose sugars like ribose for nucleic acid synthesis. This increases DNA synthesis. There is also consequent increase in protein synthesis. The tumour cells can slurp up fructose. Tumour cells utilise fructose for proliferation. The fetal cells like tumour cells also utilize fructose for proliferation. Fructose can promote metastatic deposits. The tumour cells use fructose differently from glucose. Cancer cells utilize fructose to support proliferation and metastasis. Fructose increases nucleic acid synthesis. Fructose can help the cancer cells to grow fast by inducing the transketolase enzyme and the pentose phosphate pathway. Fructose administration increases redox stress, DNA damage and cell inflammation all contributing to oncogenesis. Fructose is the most abundant sugar in the fetal tissues and is important in the development of fetus by promoting cell proliferation. Fructose is 20-times more concentrated in the fetal blood than glucose. Sperm cells and ova also use fructose for metabolism and energy. Thus all rapidly proliferating cells - cancer cells, fetal cells and reproductive cells depends upon fructolysis. Fructose is the principal diet of the cancer cells. Global warming and archaeal growth results in HIF

alpha induction. HIF alpha induces tumour growth. HIF alpha also increases glycolysis. But archaeal induced HIF alpha also induces aldose reductase which converts glucose to fructose and metabolism proceeds along the fructolytic pathway. Fructosylation of glycolytic enzymes brings glycolysis to a halt. Fructosylation of mitochondrial PT pore hexokinase can result in PT pore dysfunction and cell proliferation. The fructolytic pathway is the principal energetic pathway for rapidly proliferating cancer cells, fetal cells and stem cells. The global warming will induce the Warburg phenotype of the fructolytic variety. This leads to an epidemic of cancer. There is an epidemic of cancer in relation to global warming. The fructolytic pathway can lead to increased DNA synthesis and RNA synthesis due to flux via the pentose phosphate pathway. The fructolytic pathway can be directed to the GABA shunt generating succinyl CoA and glycine. These are substrates for porphyrin templates to form RNA viroids. The archaeal induced redox stress can induce endogenous HERV expression and reverse transcriptase expression. The RNA viroids are converted by HERV reverse transcriptase to corresponding DNA and integrated into the genome by HERV integrase. The integrated RNA viroid related DNA can function as jumping genes producing genomic plasticity and genomic change.

Fructose as said before induces the thiamine dependent transketolase flux. It increases both the oxidative and non oxidative pentose phosphate pathway. This increases nucleic acids and glycosaminoglycan synthesis. Fructose is converted to fructose 1-phosphate which is acted upon by aldolase B converting it into glyceraldehyde and dihydroxy acetone phosphate. Glyceraldehyde is converted glyceraldehyde 3-phosphate by triokinase. DHAP can be converted to glyceraldehyde 3-phosphate by the enzyme triose phosphate isomerase. Glyceraldehyde 3-phosphate can be converted to pyruvate. This pyruvate can be channeled to gluconeogenesis and glycogen storage by the action of the enzyme pyruvate carboxylase. This results in the conversion of glyceraldehyde

3-phosphate to pyruvate and via pyruvate carboxylase to glucose 1-phosphate. Glucose 1-phosphate is converted to glycogen polymers. Thus fructolysis results in glycogen storage. The pyruvate that is generated by fructolysis is converted to glutamate which can enter the GABA shunt pathway. The GABA shunt pathway generates glycine and succinyl CoA which are substrates for ALA synthesis. Thus fructolysis stimulates porphyrin synthesis. The porphyrins can self organize to form supramolecular arrays called porphyrions. Porphyrions can self replicate by using other porphyrions as templates. Porphyrions can have energetic and ATP synthesis by electron or photon transport. Porphyrions are dipolar molecules and in the setting of digoxin induced membrane sodium potassium ATPase inhibition can generate a pumped phonon system induced quantal state and quantal perception. They can function as quantal computers with information storage. The porphyrions are basic self replicating living structures. The porphyrins can act as a template for the formation RNA, DNA and proteins. The RNA viroids, the DNA viroids and proteins generated by abiogenesis on porphyrin templates can self organize to form primitive archaea. The archaea are thus capable of abiogenic replication on porphyrin templates. The archaea can induce HIF alpha and further aldose reductase induction promoting fructolysis.

Fructose is an addictive substance. Fructose affects the hedonic centres in the brain concerned with pleasure and reward. In the addiction scale fructose is more addictive then cocaine and cannabis. Fructose decreases BDNF. Low BDNF produces changes in the brain resulting in schizophrenia and depression. Fructose can also produce chronic inflammation involved in schizophrenia. The fructolytic pathway is important in the genesis of psychiatric disorders. The increased fructolysis can lead to fructosylation of lipoproteins especially apoprotein E and apoprotein B. Apo B can undergo lysine fructosylation leading to defective LDL and cholesterol uptake by the brain. This results in

autism and schizophrenia. Fructolysis leads to cholesterol depletion of the brain. Cholesterol is required for the formation of synaptic connections and cerebral cortex. This leads to cerebral cortical atrophy and cerebellar dominance in the presence of cholesterol depletion. This can contribute to the genesis of the cerebellar cognitive affective syndrome, the basis of schizophrenia and autism. There is an epidemic of schizophrenia and autism correlating with global warming. Fructosylation of LDL and brain cholesterol depletion can lead to dysfunction in synaptic transport. There is more release of glutamate into the synaptic from the presynaptic neuron consequent to a presynaptic neuron membrane dysfunction as a result of cholesterol depletion. This contributes to glutamate excitotoxicity. Glutame excitotoxicity can contribute to neuronal degeneration. Fructose can also produce zinc deficiency. Increased fructose intake produces zinc depletion leading to defective formation of metallothionines leading to defective heavy metal excretion. This leads to mercury, cadmium and aluminium toxicity in the brain leading to psychiatric disorders like autism and degenerations like Alzheimer's disease. Zinc deficiency consequent to fructose excess can lead to copper excess. The zinc containing neurons in the cerebral cortex are called the gluzinergic neurons. The cerebral cortex especially the prefrontal cortex will atrophy producing cerebellar and brain stem dominance. Copper is required for the dominance of subcortical cognitive structures. Fructose ingestion can also lead to calcium deficiency which can produce defective calcium signaling. Fructose ingestion leads to fructolysis and the generation of reactive species 3-deoxyglucosone important in mallard reachion and fructosylation of neuronal proteins leading to their defective function. Neuropsychiatric disorders and neurodegenerative disorders can be described as fructose diseases. Topiramate a fructose analogue is used to treat motor neuron disease. Fructose biphosphate aldolase B mutation has been seen in schizophrenia, bipolar disorders and

depression. 6-phosphofructo 2-kinase and fructose 2,6-biphosphotase abnormalities have been seen in schizophrenia. Fructose metabolism abnormalities have been noted in schizophrenia, manic depressive psychosis and autism. Fructose inhibits brain plasticity. Fructose inhibits the ability of neurons to communicate with each other. The wiring and re-wiring of neurons is inhibited. Fructose leads to a neuronal disconnection syndrome.

Fructose can increase flux via the pentose phosphate pathway and hexosamine pathway leading to glycosaminoglycan synthesis. Glycosaminoglycan accumulation in the tissues can produce mucopolysaccharidosis and fibrosis. Increased heparan sulphate accumulation in the brain leads to formation of amyloids plaques and Alzheimer's disease. Connective tissue accumulation in the lung leads to interstitial lung disease, in the kidneys it produces tubular atrophy and a chronic renal failure similar to meso-American nephropathy. Connective tissue accumulation in the heart can lead to a restrictive cardiomyopathy. Accumulation of GAG especially hyaluronic acid in bones and joints leads to osteoarthritis and spondylosis. GAG accumulation in the endocrine organs can produce thyroid dysfunction resulting in MNG and thyroiditis, pancreatic dysfunction producing chronic calcific pancreatitis and adrenal dysfunction producing hypoadrenalism. Accumulation of GAG in the vascular tissues can result in mucoid angiopathy contributing to coronary artery disease and stroke. The accumulation of lipids due to the fructolytic pathway along with glycosaminoglycans can lead to fatty liver. This can later lead onto cirrhosis of the liver. Fructose is the principal culprit for fatty liver and cirrhosis. The glycine synthesized from the fructolytic intermediate phosphoglycerate can play a role inhibiting fatty liver. There is an epidemic of chronic renal failure due to tubular fibrosis, mucoid angiopathic vascular diseases, cardiomyopathy, multiple endocrine failures, cirrhosis of the liver, interstitial lung disease, degenerative bone and joint diseases and degenerative

brain disease like Alzheimer's disease and Parkinson's disease as a consequence of global warming.

The increasing growth of archaea results in increased secretion of archaeal RNA viroids. They can interrupt mRNA function and dysregulates cell metabolism. This is by the mechanism of mRNA blockade. The viroidal RNA can combine with proteins generating prion proteins. This produces a protein conformation defect. This produces a prion protein disease. Abnormal protein conformation of beta amyloid, alpha synuclein, ribonuceloproteins, islet associated amyloid polypeptide and tumour suppressor protein can lead to an epidemic of Alzheimer's disease due to beta amyloid accumulation, alpha synuclein accumulation producing Parkinson's disease, prion like ribonucleoproteins producing motor neuron disease, metabolic syndrome x due to defective insulin seretion as a result of IAPP and abnormal prion like tumour suppressor protein producing tumours. These prion diseases induced by archaeal RNA viroids are also transmissible. Thus global warming related fructolysis leads to archaeal induced RNA viroidal mediated prion disease and amyloidosis. This raises the spectacle of a Cassandra syndrome of human extinction.

Fructose is phosphorylated to fructose 1-phosphate by ketohexokinase C or fructokinase. Fructose 1-phosphate is converted to glyceraldehyde which is then converted to glyceraldehyde 3-phosphate and dihydroxy acetone phosphate (DHAP). Fructose 1-phosphate is cleaved to DHAP and glyceraldehyde 3-phosphate. DHAP can enter the glycolytic pathway or can go to gluconeogenetic pathway. DHAP generated from fructose 1-phosphate by the action of aldolase B is acted upon by triose phosphate isomerase converting it into glyceraldehyde 3-phosphate. Glyceraldehyde 3-phosphate can be fructolysed to pyruvate and acetyl CoA. Acetyl CoA can be used for cholesterol synthesis for storage. The pyruvate generated from glyceraldehydes 3-phosphate can be converted to the citrate which can be used for fatty acid

synthesis by the action of enzymes acetyl CoA carboxylase, fatty acid synthase and malonate dehydrogenase. Glyceraldehyde is acted upon by alcohol dehydrogenase which converts it into glycerol. Glycerol is acted upon by glycerolkinase converting it into glycerol phosphate used for phosphoglyceride and triglyceride synthesis. Glyceraldehyde can also be acted upon by triokinase converting it into glyceraldehyde 3-phosphate which is then converted to DHAP by triose phosphate isomerase. Glyceral phosphate and dihydroxy acetone phosphate are interconvertible by the action of the enzyme glycerol phosphate dehydrogenase. Glycerol and fatty acids generated by fructolysis contribute to lipid synthesis and fat is stored. Fructose does not increase insulin secretion and doesn't need insulin for transport into the cell. Fructose is transported by the fructose transporter GLUT-5. Ketohexokinase C is exclusively seen in the liver which is the principal site of fructose metabolism. In the presence of hypoxia and anerobic states, there is induction of HIF alpha which can induce ketohexokinase C or fructokinase in the liver, kidney, gastrointestinal tract, brain and heart. Fructose 1-phosphate by-passes the enzyme phosphofructokinase which is the key regulatory enzyme the glycolytic pathway. Phosphofructokinase is inhibited by ATP and citrate. Thus stress induced fructolysis is an unregulated pathway not amenable to metabolic switches. Fructose does not depend upon insulin for its transport and fructolysis. Therefore fructolysis is not under insulin or endocrine control. It is an unregulated pathway.

The phosphorylation of fructose depletes the cell of ATP. Ketohexokinases preferentially phosphorylate fructose over glucose if it is available. In the presence of redox stress, osmotic stress and archaea/viroids aldose reductase is induced converting all the glucose to fructose. Glycolytic pathway comes to a halt as no ATP is available for phosphorylation of glucose and glucose as such gets converted to fructose. The fructose phosphorylation depletes the cell of ATP. ATP is converted to ADP and AMP which is deaminated to produce uric

acid. Fructose increases flux in the pentose phosphate pathway increasing nucleic acid synthesis. Purine degradation results in hyperuricemia. Thus fructolysis results in increase in uric acid accumulation in the body. Uric acid will suppress the mitochondrial oxidative phosphorylation as well as produce endothelial dysfunction. The depletion of ATP by fructose phosphorylation results in membrane sodium potassium ATPase inhibition. This results in reduced energy needs of the cell as 80% of the ATP generated by metabolism is used for maintaining the sodium potassium pump. This results in membrane ATPase inhibition generated hibernatory state. The glyceraldehyde 3-phosphate generated by fructolysis can be converted to the pyruvate and acetyl CoA used for cholesterol synthesis. The cholesterol that is synthesized is used for digoxin synthesis. Digoxin also has got aglycone part which contains sugars like digitoxose and rhamnose. Digitoxose and rhamnose are generated by the fructose induced flux and upgradation of the pentose phosphate pathway. Thus fructolysis results in a hyperdigoxinemic state and membrane sodium potassium ATPase inhibition. This results in cell protection and hibernation.

Fructose produces flux along the pentose phosphate pathway and hexosamine pathway. This results in GAG and nucleic acid synthesis. Fructose is converted to fructose 1-phosphate which is then converted to ribulose 5-phosphate. Ribulose 5-phosphate is acted upon by an isomerase converting it into xylulose 5-phosphate and ribose 5-phosphate. Xylulose 5-phosphate and ribose 5-phosphate interact to produce glyceraldehydes 3-phosphate and sedoheptulose 7-phosphate which is then converted to fructose 6-phosphate and erythrose 4-phosphate. The pentose phosphate pathway generates ribose for nucleic acid synthesis. The pathway also generates hexosamines for GAG synthesis. The pentose phosphate pathway also produces digitoxose and rhamnose for digoxin synthesis.

The global warming results in endosymbiotic archaeal growth. Archaea can induce aldose reductase which converts glucose to fructose. Fructolysis promotes flux along the pentose phosphate pathway generating nucleic acids and glycosaminoglycans. Fructolysis also generates glyceraldehydes 3-phosphate and further pyruvate. The pyruvate can enter the pyruvate carboxylase scheme generating gluconeogenesis and glycogen synthesis. Thus fructolysis can produce glycogen storage. Pyruvate can be converted to citrate for lipid synthesis. Pyruvate can also be converted to acetyl CoA for cholesterol synthesis. The flux along the pentose phosphate pathway generates the digoxin sugars, digitoxose and rhamnose. Cholesterol can be converted to digoxin producing a hyperdigoxinemic state. Digoxin produces membrane sodium potassium ATPase inhibition. The selective phosphorylation of fructose by fructokinase depletes the cell of ATP producing membrane sodium potassium ATPase inhibition. This results in the generation of a hibernatory state. The fructolysis generated pyruvate can get converted to glutamate which can enter the GABA shunt pathway producing succinyl CoA and glycine for porphyrin synthesis. Porphyrins can form self replicating porphyrions or act as a template for the formation of RNA viroids, DNA viroids and prions which can symbiose to form archaea. Thus the archaea are capable of self replicating on porphyrin templates. The fructolysis thus produces a hibernatory syndrome with fat, glycogen and nucleic acid synthesis and storage. Fructolysis results in the generation of a hibernatory species, the homo neanderthalis. The fructolysis generated membrane sodium potassium ATPase inhibition results in cell hibernation and ATP sparing. The lack of ATP and digoxin induced membrane sodium potassium ATPase inhibition results in cortical inhibition and cerebellar dominance. This produces a somnolent state and a cerebellar cognitive affective disorder. The porphyrions generated by fructolysis produces quantal perception and cerebellar dominance. The storage of glycogen, fat and GAG results in

obesity. The cerebellar cognitive affective syndrome results in a hypersexual state. The fructolysis and fructose can activate NFKB producing immune activation. The fructosylation of glycolytic and mitochondrial proteins suppresses the body's normal energetic which depends upon glycolysis and mitochondrial oxidative phosphorylation. Fructosylation of proteins results in blockade of glycolysis and mitochondrial oxidative phosphorylation. The body's energy needs are produced by fructolysis, porphyrin array mediated electron transport chain and ATP synthesis as well as membrane sodium potassium ATPase inhibition relation ATP synthesis. This produces a new species by archaeal symbiosis consequent to global warming - the homo neanderthalis. This can be called as the tropical hibernatory syndrome consequent to global warming.

This can be called also as a fructose disease. Endosymbiotic archaea and viroids induce aldose reductase and converts body glucose to fructose leading to preferential fructose phosphorylation by ketohexokinase C. Fructolysis results in fructose 1-phosphate being acted upon by aldolase B resulting in the formation of glyceraldehyde and dihydroxy acetone phosphate. Glyceraldehyde can be converted to glyceraldehyde 3-phosphate and this contributes to pyruvate formation. Pyruvate enters the GABA shunt resulting in the formation of succinyl CoA and glycine. They are substrates for porphyrin synthesis and porphyrion formation. The porphyrins form a template for the formation of RNA viroids, DNA viroids, prions, isoprenoids and polysaccharides. They can symbiose together to form primitive archaea. The archaea can further induce HIF alpha, aldose reductose and fructolysis resulting in further porphyrinogenesis and archaeal self replication. The archaea by methanogenesis contributes to global warming which leads to further archaeal growth and a vicious cycle with no regulatory switches. The fructolytic pathway induced by archaea by-passes regulatory enzyme phosphofructokinase and is practically unregulated.

Fructolytic pathway contributes to glycogen, lipids, cholesterol, hexose sugars and mucopolysaccharides synthesis and storage. This leads onto a hibernatory state and archaeal symbiosis induced species change resulting in neanderthalisation of the homo sapien species. The digoxin and fructose phosphorylation induced ATP depletion leads to membrane sodium potassium ATPase inhibition, sparing of ATP and tissue hibernation as most of the energy needs of the body are for the working of the sodium potassium pump. The cholesterol that is synthesized by fructolysis is catabolized cholesterol oxidases for archaeal energetics. Archaea also derives its energy from a primitive form of electron transport chain functioning in self replicating porphyrin arrays. The archaeal digoxin induced sodium potassium ATPase inhibition can lead to membrane ATP synthesis. The archaea and the new human species phenotype derive its energy from the above mentioned mechanism. The glycolytic enzymes and the mitochondrial PT pore hexokinase are fructosylated making them dysfunction. The fructosylated glycolytic enzymes lead to generation of antiglycolytic enzyme antibodies and disease states. The human body's principal method of energetics tissue glycolysis and oxidative phosphorylation comes to a grinding halt. The human body is taken over by the overgrowth of endosymbiotic archaea and assumes hibernatory state with accumulation of glycogen, lipids, mucopolysaccharides and nucleic acids. The catabolic pathways for energy generation related to glucose, glycolysis and oxphos scheme stops. The human body can depend upon ketogenesis from fat and proteins. The upregulated fructolytic pathway generates phosphoglycerate which converted to phosphoserine and glycine. They can be converted to other amino acids and used for ketogenesis. The body assumes a high BMI index and obesity with visceral fat storage and adiposity akin to the Neanderthal metabolic phenotype. Digoxin induced membrane sodium potassium ATPase inhibition results in cortical dysfunction. The brain porphyrins can form a quantal pumped phonon system

resulting in quantal perception and low level EMF absorption. This leads to prefrontal cortex atrophy and cerebellar dominance. Fructose itself leads to sympathetic hyperactivity and parasympathetic blockade. This leads onto a functional form of cerebellar cognition and quantal perception resulting in a new brain phenotype. The cerebellar cognitive syndrome leads to a robotic human phenotype. The phenotype is impulsive, has extrasensory perception and has less of speech production. Communication is by symbolic acts. The cerebellar phenotype doesn't have a cortical control and contributes to surrealistic behavior patterns. This produces impulsive behavior and an epidemic of surrealism where the rational prefrontal cortex becomes extinct. This leads to extremes of spirituality, violent and terroristic behavior and hypersexual states contributing to a state of transcendence underlined and reinforced by quantal perception. Cerebellar phenotype owing to its quantal perception behaves as a community and not as an individual. This creates new social and psychological phenotypes. Fructose induces NFKB and immune activation. This results in an immune activatory phenotype. Cultured T-reg cells on high fructose diet have 62% less IL-40 secretion than controls. This results in a hyperimmune state with fructosylated proteins acting as antigens. The fructolytic pathway can lead to increased DNA synthesis and RNA synthesis due to flux via the pentose phosphate pathway. The fructolytic pathway can be directed to the GABA shunt generating succinyl CoA and glycine. These are substrates for porphyrin templates to form RNA viroids. The archaeal induced redox stress can induce endogenous HERV expression and reverse transcriptase expression. The RNA viroids are converted by HERV reverse transcriptase to corresponding DNA and integrated into the genome by HERV integrase. The integrated RNA viroid related DNA can function as jumping genes producing genomic plasticity and genomic change. This produces a new genotype. Fructosylation of body proteins and enzymes results in a protein processing defect resulting in loss of protein

function. The human cell function due to protein fructosylation, protein processing defects and protein conformational defects comes to a grinding halt. Fructolytic pathway generates porphyrin arrays induced ATP production, membrane sodium potassium ATPase inhibition induced ATP synthesis and fructolysis induced ATP generation. This provides energy for porphyrin template induced archaeal replication. The digoxin and fructose phosphorylation induced ATP depletion produces cell membrane sodium potassium ATPase inhibition and a hibernatory state. This leads onto a somnolent sleepy state. The cholesterol catabolism by cholesterol oxidases for archaeal energetics leads to defective sex hormone synthesis. This leads onto an asexual androgynous state. The cerebellar cognitive syndrome due to prefrontal cortical atrophy consequent to porphyrion induced low level EMF perception produces a hypersexual state. This results in male-female equidominance and changes in sexual behavior of the population. Thus the fructose disease consequent to global warming results in a new neuronal, immune, metabolic, sexual and social phenotype. The human body is converted to a zombie for the global warming related endosymbiotic archaea to thrive. The neuronal, metabolic, sexual and social phenotype creates the necessary environment endosymbiotic archaeal multiplication and the human body is converted to a zombie phenotype. This can be called as a hibernatory zombie syndrome. Due to the new sexual and social phenotype with asexuality and hypersexuality and female-male equidominance the human population falls. The global warming and archaeal induction of HIF alpha resulting in the Warburg phenotype leads to changes in the metabolic scheme of the cells producing body cell transformation to stem cells. The stem cells depend upon glycolysis or fructolysis for energy needs. The Warburg phenotype produces an acidic pH which can result in conversion of body cells to stem cells. The stem cells conversion results in loss of tissue function. The cerebral cortex synaptic connectivity is lost and becomes dysfunction leading to subcortical cerebellar

dominance. The immune stem cells proliferate producing an autoimmune disease. The various tissue cells the specialized function like neuron, nephron and muscle cell all because of stem cell conversion becomes dysfunctional. This produces a stem cell syndrome with human somatic cells being converted to stem cells with loss of function and uncontrolled proliferation. The fructosylation of proteins results in protein function defects. The fructosylation of LDL results in defective cholesterol transport to the cells. This results in steroidal hormone synthesis defects. Cholesterol is required for formation of synaptic connectivity and this leads to cerebral cortical dysfunction. The hemoglobin becomes fructosylated and oxygen transport is affected. This leads to hypoxia and anerobic states. The hypoxia and anaerobic states induces HIF alpha and the Warburg fructolytic phenotype. The HIF alpha also induces aldose reductase converting glucose to fructose and inducing the fructolytic scheme. The fructolysis induced GABA shunt pathway and porphyrin synthesis results in further archaeal porphyrin template related replication. This results in further archaeal induced fructolysis and the vicious irreversible cycle proceeds. The uncontrolled growth of archaea leads to still further global warming. The world of endosymbiotic eternal archaea takes over and persists during the extremophilic climatic changes of global warming. The human beings exist as neanderthalic zombies serving archaeal multiplication. The homo sapiens gets converted to a new phenotype, genotype, immunotype, metabolonomic type and brain type. This is called as hibernatory zombie related to global warming - homo neoneanderthalis.

Table 1

	Serum fructose		Serum fructokinase		Aldolase B		Total GAG	
	Mean	**± SD**	**Mean**	**± SD**	**Mean**	**± SD**	**Mean**	**± SD**
Normal	2.50	0.195	8.5	0.405	3.50	1.304	3.50	0.707
Sy X	21.20	5.201	18.91	2.942	8.01	1.244	18.46	4.623
CAD	31.40	3.212	21.18	2.267	9.02	0.667	21.41	1.653
CVA	29.98	4.002	24.96	3.829	11.72	1.397	21.65	2.755
DCM/EMF	32.04	4.955	21.37	2.050	10.89	1.344	20.12	2.855
Tumour	27.94	3.732	22.29	1.237	9.46	1.386	20.89	1.651
Schizo	31.14	4.446	22.19	2.634	11.63	3.081	21.50	1.714
Autism	28.66	5.089	24.09	2.146	12.30	1.621	22.60	3.054
AD	33.13	2.754	19.87	1.646	11.37	1.406	22.97	3.662
PD	30.24	4.551	22.72	1.955	11.93	2.999	20.13	1.507
MS	29.88	5.150	22.29	1.641	10.87	1.895	23.47	2.878
Lupus	33.11	4.509	20.24	1.639	11.59	0.767	20.62	3.504
CRF	30.24	3.209	22.52	3.196	11.76	1.596	20.55	2.164
ILD	32.04	5.295	22.37	1.585	11.84	0.963	21.49	1.544
COPD	26.68	4.266	21.78	2.253	10.62	1.703	22.84	2.965
BA	33.59	3.938	22.45	2.472	11.30	0.783	23.50	3.225
Cirrhosis	32.53	6.737	23.00	1.722	10.49	1.373	20.57	1.878
IBD	31.75	5.236	21.89	2.292	11.63	1.304	22.46	4.030
MAO	31.53	4.507	22.07	2.324	11.32	1.343	23.89	2.936
IBS	29.90	4.299	22.52	1.995	10.93	1.498	22.09	2.797
PUD	32.49	6.487	21.89	3.431	10.85	1.606	25.27	3.693
EMF	30.79	4.740	21.47	3.056	11.65	1.427	20.54	2.192
CCP	31.16	3.635	22.42	3.126	10.49	1.476	17.94	2.276
MNG	32.24	5.864	20.46	2.864	9.82	1.135	21.42	2.662
Muc ANG	30.40	6.405	23.30	4.089	11.08	1.360	22.16	3.543
DBJD	33.06	5.970	22.42	3.714	11.21	1.660	17.76	3.556
Spondylosis	32.70	4.430	21.92	1.840	14.10	2.423	26.80	3.679
F value	17.373		13.973		13.903		21.081	
p value	< 0.01		< 0.01		< 0.01		< 0.01	

Table 2

	Total TG		**Serum ATP levels**		**Uric acid**		**Anti-aldolase**	
	Mean	**± SD**	**Mean**	**± SD**	**Mean**	**± SD**	**Mean**	**± SD**
Normal	124.00	3.688	2.50	0.405	5.70	0.369	7.50	1.704
Sy X	262.40	32.790	0.82	0.143	6.21	0.452	2.20	0.583
CAD	252.44	35.388	0.85	0.085	9.00	0.485	2.23	0.567
CVA	297.64	36.410	0.79	0.081	9.34	1.641	2.02	0.303
DCM/EMF	302.00	25.166	0.77	0.151	9.26	1.048	1.41	0.310
Tumour	277.60	34.613	0.80	0.136	7.88	0.847	1.45	0.415
Schizo	244.00	31.383	0.72	0.102	8.65	0.701	1.35	0.319
Autism	284.30	19.743	0.87	0.072	8.14	0.538	1.35	0.218
AD	244.70	22.106	0.82	0.121	8.74	0.687	1.70	0.361
PD	284.30	19.945	0.83	0.090	8.90	0.579	2.03	0.232
MS	289.89	23.406	0.74	0.115	9.59	0.783	1.80	0.402
Lupus	294.00	39.903	0.78	0.161	8.34	0.712	1.81	0.691
CRF	272.10	31.057	0.86	0.101	7.76	0.798	1.67	0.363
ILD	292.10	26.337	0.78	0.135	8.40	0.442	1.72	0.360
COPD	306.40	24.419	0.74	0.136	9.62	0.952	1.63	0.440
BA	293.80	31.555	0.72	0.134	9.51	1.059	2.10	0.572
Cirrhosis	271.80	37.818	0.79	0.150	8.12	0.747	1.67	0.377
IBD	287.50	20.414	0.77	0.102	9.44	0.924	1.30	0.223
MAO	316.20	31.283	0.76	0.103	9.32	0.864	1.41	0.307
IBS	279.10	27.606	0.77	0.095	9.68	1.060	1.44	0.350
PUD	285.70	22.628	0.76	0.126	9.77	0.957	1.14	0.134
EMF	270.10	28.792	0.81	0.079	8.76	0.881	1.31	0.329
CCP	293.00	28.111	0.78	0.145	8.30	0.966	1.31	0.265
MNG	262.70	30.324	0.83	0.091	8.04	0.667	1.55	0.493
Muc ANG	275.40	30.351	0.77	0.138	8.83	0.633	1.47	0.466
DBJD	282.60	27.573	0.79	0.136	8.28	0.978	1.89	0.315
Spondylosis	295.30	16.600	0.72	0.108	10.21	1.310	1.54	0.377
F value	16.378		59.169		14.166		55.173	
p value	< 0.01		< 0.01		< 0.01		< 0.01	

Table 3

	Anti-enolase		Anti-pyruvatekinase		Anti-GAPDH	
	Mean	**± SD**	**Mean**	**± SD**	**Mean**	**± SD**
Normal	1.50	0.358	50.40	5.960	5.20	0.363
Sy X	0.51	0.185	17.04	3.556	1.73	0.371
CAD	0.55	0.154	16.06	6.811	1.78	0.349
CVA	0.66	0.182	21.79	4.567	1.50	0.307
DCM/EMF	0.49	0.197	18.68	4.585	1.54	0.471
Tumour	0.42	0.182	19.93	2.421	1.39	0.253
Schizo	0.40	0.142	22.02	11.954	1.31	0.235
Autism	0.20	0.060	19.27	2.201	1.20	0.205
AD	0.38	0.205	18.87	3.899	1.37	0.305
PD	0.42	0.208	20.11	3.220	1.44	0.342
MS	0.39	0.124	18.93	6.447	1.78	0.355
Lupus	0.42	0.116	18.59	3.721	1.48	0.258
CRF	0.55	0.220	17.06	3.449	1.32	0.358
ILD	0.52	0.202	18.80	3.221	1.41	0.355
COPD	0.59	0.159	18.14	3.500	1.71	0.509
BA	0.36	0.177	15.33	3.212	1.72	0.277
Cirrhosis	0.48	0.273	18.60	2.915	1.52	0.287
IBD	0.43	0.163	17.06	4.366	1.40	0.298
MAO	0.44	0.230	19.08	3.396	1.48	0.220
IBS	0.57	0.242	19.99	2.637	1.39	0.289
PUD	0.51	0.221	20.63	5.116	1.42	0.329
EMF	0.42	0.182	14.55	3.133	1.24	0.239
CCP	0.50	0.149	17.82	2.889	1.44	0.234
MNG	0.47	0.151	17.59	2.469	1.44	0.270
Muc ANG	0.36	0.114	18.63	3.147	1.48	0.271
DBJD	0.54	0.211	22.48	4.638	1.33	0.302
Spondylosis	0.40	0.134	19.91	5.099	1.49	0.282
F value	14.091		21.073		58.769	
p value	< 0.01		< 0.01		< 0.01	

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